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The Jackson Lecture.[1]

SOME DEBTS OF MEDICINE TO THE FIGHTING SERVICES.

By S. F. McDONALD, M.D. (Melbourne), F.R.C.P. (London), F.R.A.C.P.,

To Ernest Sandford Jackson, in whose name this annual address is given, the study of Queensland's early medical history was a true labour of love. We remember his addresses on the early hospitals of Brisbane and on the early worthies of those days—Ballow, Bancroft, Cannon, Hobbs and O'Doherty—some of whose portraits you may see on these walls.

Now it is even possible, though statements are uncertain, that this building was occupied by the Dr. Hobbs who filled such an important place in early Queensland. Later than the days when young Tom Petrie hunted possums on Wickham Terrace with his black friends, this home was built, some say by Hobbs himself.

To Dr. Jackson all was grist to his medical history—soldiers, sailors, wounds, disease, dietetics and even transport—so that I feel he would have approved my presenting some aspects of military medical history, the more so as he and his family were among the earliest of Australian troops to go abroad in 1914.

The Debt of Medical Science to the Fighting Services.

It is a platitude that certain industries and sciences have been much developed by the needs of war—flying, explosives, metallurgy and dye chemistry, for instance. It will perhaps be a surprise to some medical men to discover how much medicine has gained from war; but the pages of military history will afford them food for reflection.

It is true that there have been occasions on which research has received little encouragement in the Service. Ronald Ross, for instance, once declared that his interest in malaria and mosquitoes caused him to be sent to a district where there were neither.

In the war of 1914-1918 the British Army was somewhat unfortunate in the officers occupying the higher ranks of the Army Medical Service. Despite the vigorous efforts of Sir Alfred Keogh as Director-General (1905-1910), for various reasons many of them were still men of little more than mediocre intelligence who, having joined the army when the Royal Army Medical Corps was very much in the doldrums, had made their way to the top by a limpet-like capacity for sticking to their positions. To such men the idea of special research during a war was an absurdity, and it was one of them who gave to a very senior consultant physician the assurance that "there's going to be no b—— research in this war", and the slogan "N.B.R." was born. But as he spoke research was going on in the most unlikely places, and all ranks were busy investigating their own problems in the new environment in which they found themselves. For instance, one morning in 1915, as the late Colonel Fred Smith was doing his rounds of inspection at Number 4 General Hospital, he was much struck by the peculiar behaviour of Lance-Corporal Evans, in charge of a "Thresh" steam disinfector. In addition to the bundles of muddy and blood-stained clothes which were being taken out, there were a number of small cardboard boxes. These were carefully opened, the contents were examined, and a note was made in a "soldier's pocket-book". Inquiry showed that the boxes which had contained living lice had been packed at varying depths among the bundles of clothes, and the effect of heat on the parasites at varying depths was being noted. It was a spontaneous piece of research that earned Evans

[1] Delivered at a meeting of the Queensland Branch of the British Medical Association on September 20, 1940.

an extra stripe and made the work of the "Thresh" disinfector much more efficient. Corporal Evans was blood-brother to McNee and his colleagues working on trench fever, to Almroth Wright attacking wound sepsis, to Dreyer studying typhoid fever and to C. J. Martin working on dysentery (to name only a few).

In fact, before the war was over there was research of all sorts going on; there was no problem which was not being tackled by some special worker or group of workers, and "N.B.R." in at least one laboratory was a slogan of work. But by research I mean not merely test-tubes and microscopes, but any original work which adds to our knowledge. Florence Nightingale planning hospitals was as much a research worker as Almroth Wright studying lymph-flow, or Captain E. M. Cowell taking blood pressure in no man's land.

In fact, all through history the fighting services have made valuable attacks on the problems of general medicine and surgery—contributions due to the zeal and enthusiasm of workers sometimes helped, sometimes discouraged, by the higher command.

I shall deal with three of these attacks on three of these problems: the attack on wounds, by the Army; on dietetics and hygiene, by the Navy; and on altitude and oxygen want, by the Air Force.

The Surgery of Wounds.

Military surgery of the pre-Lister days may be linked with the names of three men and one woman: Paré, Larrey, John Hunter and Florence Nightingale.

I suppose everybody has heard of Paré's great aphorism: "I dressed the wound, God cured it." My old teacher, the late Mr. Hamilton Russell, told me once that every young surgeon should include that among his half-dozen basic principles. That it is as true today as it was then may be seen in the success of Dr. Trueta's method of treating gunshot wounds, a method essentially the same as that of Paré's, which entailed putting the wounded limb in the best possible state and leaving the rest to the body's own power of resistance.

Another point in which Paré approached the modern attitude was the importance he ascribed to comfort and amusement during convalescence. It was, for instance, remarkable in the last war how much more rapidly men recovered who convalesced in the happy and pleasant atmosphere of England or southern France than did those retained in the boring and depressing areas of Salonika or even Egypt.

It was, of course, for his use of the ligature to control hæmorrhage instead of the searing-iron that Paré earned his greatest claim to the gratitude of the wounded soldier. True that it was still 300 years to Lister whose ligatures could be tied, buried and forgotten; true that the dangers of secondary hæmorrhage and infection along the hanging ligatures were to persist seventy years after even Nelson's amputation; but even so the advance was the greatest single life-saving step for many centuries.

Not least of the debts which war surgery owes to this great man was the recognition of the medical officer's right to criticize military measures which were resulting in unnecessary losses and suffering. Not more courage had Major Carter in Mesopotamia when he exposed the miseries of the sick and wounded after Kut, than had Paré when he rebuked the Archbishop of Lyons (at that time "G.O.C. Paris") for the sufferings of the sick, poor and wounded during the siege of the city in 1590.

No doubt many of the higher administration in Mesopotamia and India might well have echoed the archbishop's words "that the good man had fairly astonished him"; and again "this was not the sort of politics he was used to hear talked, and that Master Paré had waked him up and made him think of many things".

Our next great figure in war surgery is John Hunter. In 1761 he went as a staff surgeon in the expedition to Belle Isle. At that period staff surgeons were all those surgeons not belonging to regiments, who were employed on the staff of a general in the field or in a general hospital or garrison. There was no common body of medical troops such as the Royal Army Medical Corps, formed after 1873. This system naturally gave rise to much dissatisfaction and friction, and inevitably Hunter reacted very badly to it. He wrote to his brother:

> I have succeeded in everything that I have attempted, but my practice in gun-shot wounds has been in a great measure different from all others, so that I have had the eyes of all the surgeons upon me, both on account of my supposed knowledge and method of treatment. My fellow creatures of the hospital are a d——d disagreeable set. The two heads are as unfit for their employment as the devil to reign in Heaven.

It has never been held that John Hunter was able to suffer fools gladly. (At this date, too, there were arguments about posts with special pay and allowances which sound strikingly modern.) But at the same time Hunter was working, thinking and planning, the result being his book "Treatise on Gunshot Wounds". In this he broke away—as he hinted in his letter—from many of the established ideas, and tried to start afresh; naturally he came as much into conflict with authority as did Larrey or Milligan.

It was singularly unfortunate for the British Army that Hunter became Inspector-general only in 1790, when ill health was already heavy on him, and his life had come to be as he said, "at the mercy of any scoundrel who chooses to infuriate me". (And there must have been many scoundrels to infuriate a conscientious and able inspector-general in 1790!) For Fortescue wrote of the condition of affairs in July, 1794:

> But the very worst department of all, was that of the hospitals, wherein the abuses were so terrible that men hardly liked to speak of them.
>
> Some kind of a medical staff was improvised out of drunken apothecaries, broken down practitioners and rogues of every description who were provided, under terms of some cheap contract—the charges of respectable medical men being deemed exorbitant.
>
> How history repeats itself!

This state of affairs was in no small degree due to the reluctance of the authorities to spend the money necessary for efficiency—just as it happened 120 years later at Kut-el-Amara.

But if John Hunter came too late into the service to be a second Keogh or Carter, at least the book he had written twenty years before on gunshot wounds had caused a medical revolution, and had become the standard guide of the military surgeon. Hunter was indeed very different from most of his colleagues in his mental searchings and probings over the problems all around him. They seemed to have been prepared to accept the theories of the day and follow the accustomed routine; Hunter, on the other hand, broke away from tradition. He himself said thirty years later that it was his war experience that "drew my attention to inflammation in general".

What were Hunter's gifts to surgery from his war experience?

1. He shattered the old superstition that in war wounds there was something obscure and different from ordinary wounds (Cuthbert-Wallace was preaching to the same text at the First Army Medical School in 1917).

2. He pointed out that they were also "contused wounds", although they had certain peculiarities due to the passage of foreign bodies through the tissues. "A part of the solids surrounding the wound is deadened and is afterwards thrown off as a slough—which prevents such wounds healing by first intention." (For this reason, today it has been found unwise to close gunshot wounds by primary suture.) He pointed out how the separation of a slough might open part of a large artery or portion of intestine. This we were to relearn with many tears in France in 1914, and in 1915 it led Milligan to apply the logical treatment which was not within Hunter's reach—"cleaning up" or *débridement* by the excision of the "deadened solids".

3. He taught yet another lesson—"the greater the velocity of the ball, the cleaner it wounds the soft parts". That, too, we were to relearn, for the experience in South

Africa with wounds made by small high-velocity bullets was misleading when applied to the wounds from blunt and slow-moving shell fragments in France.

Another matter we might have learned from him was that these more slowly moving fragments had often a less straight track, so that the position of the foreign body was less easily ascertained.

But his greatest lesson was that it was wrong to interfere with any wound unless there was a definite object to be attained. By his influence and reputation he was able to check the practice of routine opening up of every wound regardless of any indication for so doing—"a universal custom before he challenged it", says Bowlby. In this, of course, before the advent of asepsis, he was even more correct than he would be today. When, as too often, the probe of the surgeon served to introduce more dirt, and his knife to infect from the preceding week's septic amputation, there was little to be gained, and Hunter and Trueta meet on common ground with Paré.

In all these observations we see the working of a keen and critical mind, and we see, too, how Hunter's interest, turned so powerfully to consider the inflammation which he saw all about him, remained the impetus which governed its activities for the rest of his days.

It was never given to Hunter to learn the cause of inflammation; his contemporaries saw little benefit to his patients from all his endless experimenting and thinking (no doubt they too laughed at "B.R."); but in those early days at Belle Isle, when he walked through the wards or breathed the pure air of the sea, he was laying the foundation of the modern scientific surgery of a hundred years later.

Owing to his ignorance of the real nature of inflammation, he inevitably made mistakes. He held, for instance, that early amputation was unsound, and that the wound should have time to settle down before operation. It was a view condemned equally by his British successor, Guthrie, and by the great Napoleonic surgeon, Larrey. Both these insisted that the amputation should be performed at the earliest possible moment; and Guthrie speaks of a surgeon's needing only his medical pannier for the operating table (or chair, rather), a clean cloth on which to spread his instruments and the open air. Larrey, as we shall see, got right up into the front line, and once in Egype he had just amputated one of General Silly's legs when the British cavalry swept down on him. How to save this patient? Larrey carried him off pick-a-back, at the same time grabbing up his priceless instruments—and got away from the cavalry.

For the most astounding figure in military surgery is this Frenchman, Jean Dominique Larrey, Napoleon's great director-general. He was far more than a mere surgeon; he made the French medical service the model on which our modern services are based, and his work was guided by two principles: (i) the evacuation and treatment of the wounded at the earliest possible moment; (ii) the complete responsibility (even to the risking of his life in combat) of the medical officer for his wounded. The two things go together as a matter of course nowadays; but it was Larrey who laid down these principles once and for all.

Not only that, but Larrey is said to have been the only man in France genuinely quite unafraid of the Emperor whom he served so well. They met first in the campaign of Italy, and Larrey never left him till the disastrous evening of Waterloo, when Napoleon's *"Sauve qui peut!"* found no echo in Larrey's heart. While Napoleon was hurrying along the dusty roads to the French frontier, Larrey was collecting his wounded until he was attacked by the Prussians, taken prisoner, beaten, robbed and led out to be shot, when a lucky recognition saved him. The story of Larrey's defence of his wounded on this occasion reminds us of incidents when British medical officers have had to take weapons and fight to save "their wounded". (For example, there was Surgeon John Crimmin, who in the Burmese War of 1889, while defending his wounded, thrust his sword through one Karen then wounded another, and so gained one of the Corps's many Victoria Crosses.)

Larrey's experience, too, reminds us how little the Prussians have altered in 120 years. In contrast we may take pleasure in the knowledge that when the Duke of Wellington learned that the queer vehicle running about among the French troops under fire of the British guns was Larrey's ambulance, he at once ordered the guns to seek other targets, then bared his head in homage to this act of gallantry.

Again, in the Paris revolution of 1830, Larrey, then an old man, had a hospital filled with French guardsmen wounded in the street-fighting. The revolutionary crowd were bitter and determined to have blood for their blood. They attacked the hospital, beat upon the doors, which were opened to reveal Larrey, who met their shouts with: "What do you want? *My* wounded? But they are mine! Go away!" They went; thus, thirty years before Dunant, he had laid down the principle that the wounded were immune from further injury, and it was the surgeon's duty to secure them this safety.

Until Larrey there was little thought for the wounded; the "ambulances" (that is, the first-aid dressing stations) were kept three miles behind the line, and the wounded men were left on the field of battle till after the fight; then they were collected at a "convenient place" to which the ambulance could be brought, often up to thirty-six hours after the actual wounds had been inflicted. Naturally the mortality rate was high and the suffering appalling.

To meet this Larrey devised his "flying ambulances" capable of working in close touch with even the most advanced units of the army. At first they were purely dressing units, staffed by three surgeons and an orderly; then came the idea of transport, and "this unit was fitted with pack saddles and baskets for the transport of the wounded. The jolting to which they were subjected led to this equipment being replaced by light and heavy carriages fitted with stretchers." By 1797 Larrey had perfected in every detail the organization of the ambulance services, to each of which was attached one surgeon-major of the first class (corresponding to our lieutenant-colonel), 14 surgeons and a large staff of orderlies. The transport comprised 12 light carriages (two-wheeled) and four of heavier construction (four-wheeled), but all exceptionally well sprung. There has been no advance in principle since.

The fourth great name in wound treatment is that of a woman—Florence Nightingale. Here the relation of military experience to civil practice is obvious. From the horrors of Scutari and her fights with the army administration, Florence Nightingale came back to the wards of Saint Thomas's Hospital. Here, not only did she rule the training of her own nurses, but from it, by voice and letter, controlled the growth of nursing and hygiene throughout the world. No more striking testimony to her personal influence can be found than in the splendid way nursing developed in Britain, while in Germany—where she had first gone to learn her nursing at Kaiserworth—it remained a backward and subordinate service.

Even in 1870 the future Empress Frederick was very bitter over the dirt, over-crowding and stuffiness of the German base hospitals, while the managing ladies seemed quite satisfied. She wrote of her own special hospital at Hamburg:

> To overcome the prejudice of doctors and patients against fresh air is really almost impossible. We have not one nurse nor dresser here yet, only people from the town who are dirty, ignorant and useless in the extreme, but we have sent for some better help.

Her work was appreciated by her husband, the Crown Prince, but by few others; as he noted in his diary at this time:

> In Hamburg she has created a perfect model hospital. I communicated to his Majesty [his own father] much of what I had learned, but without hearing one word of commendation in reply.

Meantime Florence Nightingale was building up at Saint Thomas's a standard of nursing which spread throughout the world and finds its flower in our present-day nursing service. It will interest some of my hearers

to know that Lucy Osburn, the first matron of the "Sydney" (the first public hospital in Australia), was one of Miss Nightingale's nurses at Saint Thomas's

What does surgery owe to these four remarkable people? To Paré the ligature, good nursing and the recognition of *vis medicatrix naturæ*; to Hunter the description of the true nature of gunshot wounds; to Larrey early operation and rapid transport; to Florence Nightingale modern nursing.

To all we owe the firmly rooted tradition that to the army nurse or surgeon the sick and wounded come first; that anything must be dared for their sake—the frown of superiors, the criticism of colleagues and the weapons of the enemy.

The Navy and Hygiene.

The life of the men of the navy differed from that of those in other walks of life by the fact that no small part of their struggles was not merely with their fellow men but with their actual way of life and with the very elements.

On active service, it is true, the soldier of, say, Marlborough's wars, had a hard enough life—billets, bivouacs, marching and fighting. Sickness was often an accompaniment aggravated by poor food, clothes and equipment. Indeed, it was in no small degree to their care in these matters that Marlborough and Wellington owed their success. Marlborough's great march that ended at Blenheim was marked by a solicitude quite unusual among contemporary army commanders for his men's health and comfort. Boots and bread, says Fortescue, were his especial care. A century later the whine from the wooden wheels of the Spanish ox-wagons that carried its supplies formed the *Leit-motif* of all the Peninsular Army's battles and marches.

But at other times the soldier lived in comparative comfort. Certainly, his barracks would have been condemned by any sanitary inspector of today; but in those days they were often better than the hovels and slums from which the recruit had come. His food might be deficient and of poor quality, but at least it was moderately fresh and he might even supplement it outside. Sanitation might be primitive and even barrack fever was not unknown; but much of his life was passed in the open air and hospitals of a sort were within reach.

It was not so aboard the King's ships. Here, packed with their hammocks touching one another, without ventilation, the air about them fœtid with filthy humanity and the reek of the bilges, lived, except for the space of their watch on deck, the seamen of England from Drake to Nelson. They were so verminous that shaved heads and wigs were for long general among the officers, and you may remember that Roderick Random, before making his first visit to the sick bay, was advised to put his wig in his pocket. Typhus was not unknown; but other contagious diseases, quite apart from the "unhealthiness" of such stations as the West Indies, were all too common.

As for the food, they had weevily biscuits, rotten pork, iron-hard beef, rancid butter, mouldy pease and stinking water; there was only one attractive article in the sailor's life, rum. Although other diseases were rife, the real terror of the sailor's life was to many a mystery for which there was no explanation—scurvy. When there were voyages of any length, sooner or later the disease appeared. The word constantly recurs in Hakluyt, and it is most likely that those 90 sick men of his little crew of 190, whom Sir Richard Grenville stayed to bring off, were suffering from scurvy.

The most terrible example of a scurvy-stricken voyage is Anson's great voyage round the world in 1740-1741. Of this, Rear-Admiral Muir, to whom I am throughout much indebted, gives an admirable account, which has all the weight of modern science and knowledge behind it. I would direct you, however, to another account written by an undeservedly forgotten Australian, W. H. Fitchett, in his book "Fights for the Flag" (*circa* 1898). Its very ignorance of the causes at work make it all the more impressive, while his account of its ravages expresses the layman's horror of the disease.

All this time, too, Anson's crew was perishing fast with scurvy of the most malignant type. The body of a poor scurvy-smitten wretch was mottled with black spots. Limbs and gums were swollen to an enormous size, and broke into putrid eruptions. Old wounds broke out afresh. Bones that had been fractured, and had set firmly again for years, parted, the new bone dissolving into mere fluid. On the body of one veteran, who had been wounded fifty years before at the battle of the Boyne, the wounds gaped blood-red, as though just inflicted. The men died so fast, or sickened so hopelessly, that by the middle of June (nine months) from the *Centurion* alone more than 200 corpses had been thrown overboard, and, to quote the chaplain's record, "we could not at last muster more than six fore-mast men in a watch capable of duty!"

There were probably, Admiral Muir suggests, at this time three diseases which took toll of the eighteenth century crews—scurvy, beriberi and typhus; but scurvy was the worst of these. Even by Elizabethan sailors it was recognized that the cause was the absence of fruit and green vegetables, and in 1753 Dr. James Lind, of Haslar Hospital, published an excellent "Treatise on Scurvy".

His was no theoretical knowledge, for in the years from 1748 to 1750 Captain Palliser, acting under his instructions, had made a voyage of fourteen months and five days "with a ship's company in perfect health—having buried only one man who died in a salivation for Pox". (Our army in the war of 1914-1918 likewise suffered fatalities in the over-zealous treatment of pox; but they were from arsenical poisoning.) Palliser was later to become Cook's friend and patron.

Later Lind wrote an essay on "The Means of Preserving the Health of Seamen". "Both these communications", Admiral Muir states, "are remarkable for their sound common sense toward hygienic problems; failing . . . only when the pathology of disease was discussed and (when) attempts were made to base treatment on that pathology rather than experience."

It is probable, as suggested before, that two other diseases were confused with scurvy, both of which were common enough—typhus and beriberi. The incidence of the former would explain why Lind considered that scurvy was more common among the gaol-birds who were so often drafted into the navy. (These men were not, like certain men of the Australian Imperial Force, given the option of joining the Australian Imperial Force or going to gaol, but were the inmates of gaols already undergoing sentence, who were simply swept up *en masse* and sent to make up vacancies in the fleet. In neither case, one feels, can the result have been happy, though Fortescue says the army was keen to have them.) Hence Lind regarded scurvy as an infectious disease and advised various means of preventing it, notably by cleanliness and disinfection (fumigation by the burning of old rags and so on was one of his methods). Now, while these would have no specific effect on scurvy, yet by a measure of "delousing" they would help against typhus.

On the other hand, Lind was most emphatic on the dietetic treatment and prevention of scurvy. He recommended eschalots, onions, leeks and garlic and a noggin made up of small beer, brandy, molasses and vinegar. While in harbour he insisted that sailors should receive fresh beef, turnips, cabbage, carrots, apples, fresh soft bread (as opposed to the hard weevily biscuit issued at sea), cider, lemons, oranges, dandelion and brook lime (water cress). At sea a "rab" (or conserve) of lemon and orange he considered should be made compulsory, though it is probable that the boiling in manufacture made this of small antiscorbutic value. He also insisted on absolute cleanliness, moderation of salt diet and unlimited fresh water. Further, he emphasized the need for fresh air and made suggestions for the maintenance of ventilation when the ship was battened down in bad weather. But in Lind's opinion nothing compared in value with the efficiency of lemon juice, an opinion which agreed with that held by seamen since the time of Drake and Hawkins.

Various auxiliary measures were suggested; for example, a Captain Latham, of the *Tygar*, considered that the remarkably healthy record of his crew was attributable to

cold sea bathing. It probably acted entirely through the resulting cleanliness.

Lind also pinned his faith to the anti-scorbutic value of a method of preserving greens by carefully washing the leaves and storing them in a cask with intervening layers of rock-salt. Cook used this method to preserve a *Sauerkraut* which he esteemed highly, and he claimed that vegetables preserved in this way lasted several months.

Against these desiderata, what at that time actually was the sailors' food? The following is a probable ration for a week: bread, seven pounds; butter, a quarter of a pound; cheese, half a pound; beef, seven pounds, or pork, four and three-quarter pounds; oatmeal, one pint; pease (dried), three and a half pints; rum, seven gills. There is nothing in that to prevent scurvy or beriberi. But this was when things went well; too often the "bread" was board-hard biscuit which contained more weevil than biscuit (it was not till just before the war of 1914-1918 that Sir Wilfred Beveridge, Director of Hygiene, worked out a weevil-proof biscuit), the butter was rancid, the meat and pork were rotten and the water was putrid. On this scene of misery and ignorance there enters James Cook, dietitian and hygienist, in his first voyage of 1768.

To begin with, his provision was very different. He had a free hand in making his choice of stores, so that everything was of the best quality. Next there was certain special measures—*Sauerkraut*, portable soup and above all malt; from the last was made "wort", a sort of infusion of the germinated barley. It was given to every man who had a sign of scurvy on him. It probably was worthless as an antiscorbutic, but its high content of vitamin *B* must have made it a specific against beriberi.

Cleanliness was another of Cook's measures which he valued highly—not only cleanliness of the men and their clothes, but also cleanliness of the whole ship.

No doubt the foregoing measures had little effect against scurvy; but they were backed up by Cook's insistence that no opportunity should be neglected of procuring fresh food; he sought meat, vegetables (even such unlikely things as wild celery in Terra del Fuego), fruit (above all, fresh citrus fruit), wine, and an especial feature, fresh water.

Cook, too, was a psychologist; to make sure that the sailor, always suspicious of new-fangled foods, should eat the *Sauerkraut*, celery and foreign fruit when provided, he ordered that they were also to be served at the officers' tables. The trick was completely successful. (To gain the same end Bligh flogged his crew.) In Cook's own words:

> We came to few places where either the art of man [that is, vegetables procured from the natives or sown by the ship's company] or the bounty of nature had not provided some sort of refreshment or other, either in the animal or vegetable way.

We must remember, too, that he took special measures against fatigue; he first introduced the eight hour day (to be followed by his less fortunate subordinate, Bligh); he protected his men from cold by special clothing (fearnought cloth, for instance, which he used, is still used in the winter suits of the navy); against cold and exposure, he had his ship's quarters warmed and aired; all were excellent measures, and the more excellent in that they allowed the body to conserve that small reserve of vitamin *C* which it lays down in times of plenty.

Cook's first voyage occupied three years; in it he had not one death from scurvy. It is little wonder that his report to the Royal Society earned him the Copley Medal for 1776, although, owing to his departure on his last voyage, he never actually received it. The only deaths from disease occurred, as a matter of fact, through Cook's very anxiety for better food. At Batavia he took in all he could of fresh fruit and vegetables. These, unfortunately, as is too commonly the case, were no doubt grown in the usual oriental sewerage irrigation system, and the result was an epidemic of severe dysentery. By the time he sailed every man had been ill and they had buried seven, including the surgeon, Moorhouse, who seems to have been a man after Cook's own heart; during the following six weeks at sea 23 more were lost.

But the work had been done; Cook had been able to show that a ship could sail the high seas for three years and lose not one man from scurvy, that improved methods of feeding, clothing and housing men could be used with but little extra cost and a great return in efficiency, but in particular that all this depended on the intelligent administration of the ship. Whether it was the work of the medical officers or of the commander, it still remained somebody's duty, and that somebody must be prepared at all times to fly in the face of custom, prejudice and service routine to secure his results.

The Air Force and the Problems of High Altitudes.

The air force is itself so young and its problems are apparently so essentially its own, that at first sight there seems little for medicine as a whole to have gained from scientific efforts to solve them. It is hard, too, for many of us to realize that we are actually seeing history made, or only finished, all within our own life time. It seems a far cry to Captain Cook, John Hunter, or even Florence Nightingale from the work of Haldane and Barcroft, Scott and Flack.

Actually the problem of high altitudes was not a new one; the Spaniards had met it in Peru when the imported negro mine workers died in hundreds even at moderate altitudes, and even latter-day miners in the Andes were to find altitude a handicap.

Although the earlier aviators worked by rule of thumb and built their machines by trial and error, yet the theory of flight had been discussed for years before; Maxim, Pilcher, Langley, Hardgrave and Lilienthal by their experiments had collected data which the first birdmen began to use when they built their planes. Then, as more and more planes were built, more and more did they become a matter of mathematics and exact scientific measurements. The stick-and-string patchwork which killed C. S. Rolls soon gave place to better things, so that when the first "Avro" was built it had become obvious that the days of hit-or-miss flying and aeroplane-building were closed.

Thus the whole atmosphere into which the Army Flying Corps was born in 1913 was a scientific and technical one. Even before the formation of the Royal Air Force, in 1917, the medical aspects of flying were engaging attention.

As the first airmen flew at only a few hundred feet, problems of altitude mattered little; airspeed was not yet great enough for blackouts to be more than hypothetical hazards, and heights of over 10,000 feet were rare during the first year of the war. But by 1917, even in 1916, the heights were increasing steadily above 10,000 feet, and soon it was found that some pilots were unable to fly at these altitudes. While most men were able to fly quite well up to 15,000 feet, over this height they began to lose concentration and even consciousness.

It had, of course, long been known that high altitudes interfered with bodily and mental activity. The classical example was the ascent of Coxwell and Glaisher in 1862 by open balloon to a height of seven miles. These navigators successfully returned and their notes provided the foundation of our modern knowledge. But over them there was a difference of opinion whether it was the low pressure or the lack of oxygen which caused the trouble.

To Paul Bert is due the first clear proof that it was the oxygen lack which was at fault, and his work was confirmed by Haldane and his co-workers, Barcroft and others since, among them the climbers of Mount Everest. The symptoms of the condition have been well described by Raymond Green, doctor of the 1933 Everest expedition, as follows:

> The appalling panting of unacclimatised men, the air starvation, the rapid pulse, the lassitude which made every step a struggle, the sleeplessness, irritability, mental deterioration, grinding headaches, mountain-sickness, and loss of appetite . . . are all the symptoms of lack of oxygen.

It must be remembered that the high altitude pilot differs from the mountaineer in that he gets little chance to become acclimatized. Green, too, points out that another

physical effect of oxygen lack is lowered resistance to cold, even to frost bite.

This introduces another danger in high altitude flying—cold. Air temperature falls 3·5° F. for each 1,000 feet, until at 36,000 feet it becomes constant at −70° F. Cold and oxygen lack are synergistic and their joint effect is to slow down metabolism, circulation and oxygen exchange, especially in the extremities, so that the danger of frost-bite is increased. This is exaggerated when there is deficient circulation from old scars and wounds.

To take a recent application, a would-be "air crew" member presented a forefinger tip distorted by a Böhler wire extension. His acceptance was for a time a matter of doubt; while another candidate's badly scarred foot, which produced apparently no trouble at ground level, did cause his rejection. It has, too, an important bearing on the dangers of circulatory instability among pilots with its accompanying cold fingers and toes.

In addition to the work on the physical side, Lowson in 1923 pointed out the very profound emotional degeneration which takes place in those exposed to low oxygen pressure. These conclusions were borne out by the mountaineers, especially by the Everest climbers, who incidentally emphasized the importance of suitable (and appetizing) food for the worker at high altitudes.

Now, it is evident that all these handicaps are the last to which a fighting pilot should be subjected. He is engaged in handling a complicated machine, moving at speeds which allow a very small margin for reaction to change; he may have to display the utmost visual and mental alertness; and worst of all, his oxygen pressure will not be nearly so constant as that of a man climbing by rail to a mountain top. He will change his altitude suddenly, diving from many thousands of feet to ground level at a rate of 600 feet a second, or climbing to 11,000 feet in under five minutes (that is, at a rate of 24 miles an hour or 40 feet a second). There is no time then for the elaborate body adjustments by corpuscle and hæmoglobin increase that Barcroft found in the Andes.

As the result of much work it can safely be said that any man who tries to fly in war time at a height of more than 15,000 feet without oxygen is not a hero, but a fool, and a disobedient fool to boot.

In addition to the problem of lowered oxygen supply, the pilot has to face two other handicaps—that of mere altitude with diminished total pressure, and that of exhaust gas poisoning.

If the last factor is omitted, as being outside this paper, the disturbance of total pressure involves mostly the accessory nasal sinus and the middle ear. All these cavities communicate with the outer air by narrow passages which may be further narrowed permanently, for example, by a distorted septum, or temporarily by a mass of infective mucopus, for example, in the course of a "cold". Trouble from either cause is more apt to occur during descent than during ascent, and the danger arises from the increasing pressure of the outer air compared with the low pressure previously set up in the cavity during ascent. Generally air can escape from the cavities more easily than it can enter; in the case of, say, the sphenoidal cells during descent, this inequality of pressure may set up a rush of air to adjust the balance; this rush will then carry infected mucopus deep into a sinus, hitherto free of infection. The same process may go on with the middle ear; but here the cavity is bounded in part by a membranous wall, so that this inrush may even cause rupture as well as set up an acute inflammation inside it. Either of these changes will rapidly reduce a pilot's efficiency. The solution of the problem, of course, lies in a thorough examination of the nose and throat of pilots, and in the correction so far as possible of any defects.

Another real though less urgent problem is the expansion of gas in the other closed cavity of the body—the alimentary canal—as the surrounding pressure falls. Fortunately it is not a great matter; but it is one reason for the insistence on a firm abdominal wall in the would-be war pilot.

But the oxygen problem is much more subtle and difficult. The obvious solution is to supply the pilot with oxygen as the natural atmospheric supply fails. Experiments to this end were begun in 1917; but the solution is by no means so easy as it sounds. In the first place, it was found that many pilots were reluctant to adopt the artificial oxygen supply. The reasons were partly psychological—a desire to "kid hardy" in colloquial language—and partly from the discomfort and irksomeness produced by any form of respirator.

Next it was found that to leave the task of turning on his own oxygen supply to the pilot's feelings, even when he was quite willing to do so, was quite impracticable. A pilot's hands in a fighting plane are already all too full, and eyes have more than enough gauges and instruments to watch, while to trust to his sensation is still worse. The most striking feature of oxygen lack is the insidiousness of onset. The sufferer, if anything, feels more fit and confident than he did at low levels. As a result it is now an order that the pilot turns on his oxygen long before it is needed, say at 10,000 feet, just as he puts on his parachute long before he needs that.

But to regulate quantity, various methods have been devised; among them was introduced in 1918 an automatic oxygen controller.

A story is told of its first trial, which may or may not be apocryphal, but is so good that one hopes it is true. A pilot was chosen for the test who was well known to suffer early from oxygen lack, and after a trial in a low-pressure chamber with complete success it was decided to make a final trial in a plane. A Bristol two-seater fighter was chosen and the designer decided to accompany the pilot. The test was a huge success, and the pilot to his surprise climbed to 20,000 feet without the slightest disturbance. In sheer delight and lightness of heart he came home with every form of roll, spin and dive known to him. He did not, however, explain this to his passenger, and though he sprang from his plane in the highest spirits, the case was far otherwise with the inventor, who had to be lifted out!

As a matter of fact the purely automatic type was soon given up and a method was devised by which the pilot turned on an increasing amount, measured by a flow meter calibrated in thousands of feet.

For very high work the combination of cold and oxygen supply became most intricately entangled, so that the oxygen itself had to be warmed before being delivered to the pilot.

It is a somewhat humbling fact that much of the experimental and practical tests for this very necessary research was carried out, not by the State, but through the munificence of a private individual in preparations for the flight over Everest. (If today our fighters have planes of the highest quality and our high-flyers an efficient oxygen supply, we owe it to the patriotism, vanity, what you will, of the eccentric Lady Houston.)

As was hinted at earlier, individuals vary greatly in their response to oxygen lack, and while some fail at anything above 10,000 feet, others are not affected till they reach 15,000 feet and a few rare spirits not till after 20,000 feet. In 1918, indeed, the Independent Air Force in "D.H. 4's" were flying at 22,000 feet. It was at this height that the attack on Berlin was to have been delivered in November, 1918. One wonders how many aeroplanes would have returned!

Therefore it soon became necessary to work out some means of testing pilots on enlistment to weed out the unsuitable types. Now, just as the Everest climbers have learned the "futility of vague generalizations about the Everest figure", so the air force has seen that such physical extremes as Kingsford Smith or Hinkler are equally possible in a first-class pilot. But there do emerge a number of simple facts, whose non-realization by the public and prospective pilots has given rise to much heart-burning and criticism. These facts are as follows:

1. A slow pulse rate at rest or under the nervous strain of the medical examination is a necessity. A pulse rate over 90 per minute is suspect, and over 100 it entails at least temporary rejection. I say temporary, if the cause

can be ascertained and removed; two outstanding pilots were rejected at their first examination because of tachycardia, in one case due to infected tonsils, in the other to excessive cigarette smoking.

2. A high blood pressure and a large pulse pressure usually disqualify a recruit. But these may be only "nervous", say the critics; agreed—but under conditions of oxygen deprivation these nervous individuals break down (McFarland) more easily than the normal; thus, at 15,000 feet, 40% of the normal adapted themselves easily, but only 15% of the "nervous" did so, and 44% of the latter class collapsed, as against 14% of the former. It is remarkable, too, that the symptoms most complained of were apathy, loss of capacity for close attention, loss of memory, judgement and self-criticism—fatal failings in a pilot or air crew.

3. Reaction to simultaneous blowing and breath-holding (the response to the well-known 40-millimetre mercury test) should be of the normal type. Here, again, it has been shown many times that the candidate giving a bad response may suffer from one of various conditions which will cause a breakdown with oxygen deprivation.

4. The Lewis exercise tolerance test should generally produce a return of the normal type. This is in itself often enough to indicate the individual's likely response to oxygen lack. It is one of the group of tests on which a final opinion is formed.

5. The late Wing-Commander Martin Flack, whose work laid the foundation of our knowledge in these problems, worked out an efficiency formula, based on the various factors which I have mentioned. It is seldom used nowadays in its original form, but his basic principles stand to this day.

Flack's work was later developed by Tredgold, and most of the principles on which the selection of candidates is based are derived from the work of these two men. Tredgold also insisted on the factor of general recognition by the examining doctor, and held that the preliminary history taking often gave more information than all the elaborate tests that could be devised.

From their work it is now generally possible to pick out, not necessarily the types that will make good pilots, but the types that certainly will not. The only sure test of a pilot is flying; no matter whether a man is physically perfect or not, there may be some subtle handicap which the aeroplane, and the aeroplane alone, can find out. But by constant research and experiment we are more and more excluding our sources of error.

And from these continued examinations and tests a mass of information is being acquired which will be helpful, not only in the study of flying and its effect on the human animal, but also a very real investigation into general physical make-up and its relation to all forms of stress and strain. Its full value we are too close and too busy as yet to be able to assess.

Conclusion.

In conclusion, I hope I have been able to convince you, not only that there is a word for medical research during war, but that it is not the same word as that used by the officer whom I mentioned in the early part of this address.

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RIEDEL'S DISEASE AND LYMPHADENOID GOITRE.

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MANY practitioners of medicine make their first acquaintance with Riedel's disease through an error in diagnosis. The large, hard, thyreoid gland, possibly adherent to other structures in the neck, at once points to a cancer of that organ, whilst the surgeon labouring through a difficult thyreoidectomy is only too willing to agree with the suggestion. Doubts about the diagnosis first arise when the gland is inspected after operation; the pathologist later recognizes it as a specimen of Riedel's disease. If more attention were paid to the symptomatology of this condition, such a sequence of events would be infrequent. Moreover, any time spent in the study of Riedel's disease and lymphadenoid goitre is well repaid by the light that is thrown on many aspects of thyreoid abnormalities. For instance, one is confronted immediately with the questions both of thyreotoxicosis and of myxœdema, the problem of metaplasia in thyreoid epithelium, the nature of large eosinophilic parathyreoid-like cells in the thyreoid gland, and the significance of lymphoid tissue in the organ. Of special interest, too, is the possible relationship between Riedel's disease and lymphadenoid goitre on the one hand and between Riedel's disease and acute or subacute thyreoiditis of known ætiology on the other.

The present account is based largely on the study of a series of 21 cases (nine of Riedel's disease and 12 of lymphadenoid goitre); but due regard has been paid to other cases recorded in the extensive literature.

Historical.

In 1883 Riedel observed a case of iron-hard "tumour" of the thyreoid gland. Twelve years later he saw a patient with a similar condition, and he saw a third in the following year. He published his observations in 1896 and 1897, named the disease "*eisenharte Strumitis*" and regarded it as a chronic inflammation.

Bowlby, in 1886, described an almost certainly similar case under the title "Infiltrating Fibroma (? Sarcoma) of the Thyroid Gland". Among the early writers to draw attention to the condition were Tailhefer, Ricard and Berry. All were impressed by the hardness of the thyreoid, the extreme fibrosis, the ease with which it could be mistaken for a malignant growth, and the involvement of cervical tissues beyond the confines of the gland. Until 1912 no mention was made of extensive lymphoid infiltration in association with the fibrosis, and the disease was generally assumed to be of a chronic inflammatory nature.

In 1912 Hashimoto described three cases of enlargement of the thyreoid in which the acini were replaced largely by lymph follicles and diffuse lymphoid tissue. He considered

that he was describing a disease that had not previously been recognized. In 1928 Ewing stated that he had studied four case of goitre, in each of which histological appearances in the same gland characteristic both of Riedel's and of Hashimoto's disease were seen. Therefore it appeared to him that Hashimoto and Riedel had described the early and late stages respectively of the same pathological process. Detailed reports were not given, but it was mentioned that in one instance the condition was first wrongly diagnosed as a carcinoma. Ewing also noticed that in the early stages of the disease the reticular cells of the lymph follicles might be so numerous as to suggest the presence of a lymphosarcoma. He considered the disease to be an infective granuloma. In 1929 Williamson and Pearse used the term "lymphadenoid goitre" in describing glands similar to or identical with those previously discussed by Hashimoto.

Between 1928 and 1931 most writers accepted Ewing's opinion on the relationship between the two syndromes, and articles appeared in the literature describing typical cases of *struma lymphomatosa* as cases of Riedel's thyreoiditis. Subsequent to 1931, some writers (McClintock and Wright, Clute, Eckerson and Warren, Gilchrist) have adhered to the view that lymphadenoid goitre and Riedel's thyreoiditis are separate and distinct diseases, while others express a belief in their essential unity (Shaw and Smith, Benson, Heyd and Poer, Vaux). This conflict is evident in the most recent contributions to the literature, Vaux supporting the unitarian concept, and Hellwig denying it. Unfortunately, Vaux has confused the issue somewhat by including in her list of 38 lymphadenoid glands many "toxic" goitres with focal areas of lymphoid tissue. All previous workers were careful to exclude this type of case when discussing the disease; but Vaux confidently asserts that it represents an early stage in the pathological process—a statement for which there is little supporting evidence.

Riedel's Disease.

Pathogenesis.

As previously stated, all cases reported as of Riedel's disease are not of the same type; some are of typical lymphadenoid goitre and others belong to the group of infective thyreoiditis secondary to infection of the upper portion of the respiratory tract. If these are excluded, the evidence suggests that in the remainder the process is chronic inflammatory in nature. The early stimulation of the gland function, followed by destruction of the parenchyma, extensive fibrosis, and the presence of cells usually found in chronic inflammatory states, and the presence of similar lesions in the adjacent cervical tissues, are in keeping with this view. The injection of chemical irritants into the thyreoid glands of animals will produce similar histological changes, but this cannot be regarded as having much bearing on the ætiology of the human disease. Specific microorganisms have not been found constantly in Riedel's thyreoiditis.

Such views as those which hold that the disease results from adrenal dysfunction or from degeneration of a previously enlarged thyreoid, lack any convincing support.

Lymphadenoid goitre is regarded by many as a metabolic disturbance producing a local effect on the thyreoid. Some who hold this condition and Riedel's disease to be identical consider that the latter also is a disorder of metabolism.

Pathological Anatomy of Riedel's Thyreoiditis.

Macroscopically the "typical" gland of Riedel's thyreoiditis differs from that of lymphadenoid goitre in that it is firmer and lighter in colour. The surface is usually moderately smooth, the hard superficial nodules sometimes found in carcinoma are absent.

In nearly one-third of reported cases the disease is localized to one lobe. In section, the opaque, hard, whitish tissue may occupy most of the gland, but considerable areas of unaffected tissue may be seen. In Meeker's case most of this was near the surface, but the distribution is very variable. These regions usually show areas of colloid storage, normal vesicles or hyperplastic epithelium. Macroscopically the appearance of the gland may be mistaken for thyreoid carcinoma, but in Riedel's disease the tissue is usually whiter and firmer to cut, and the surface is less irregular. Islands of carcinoma cells are absent. The pretracheal fascial capsule is often thickened, and the fibrous tissue may extend beyond the gland to other structures in the neck. Extraglandular fibrosis may reach from the base of the skull to the mediastinum, causing constriction of the œsophagus, the carotid vessels, the internal jugular vein, and the cervical nerves. Fibrosis may be found even in the subcutaneous tissues, giving an easily palpable hard thickening. Ulceration of the skin has been recorded (Joll). The trachea and œsophagus, in addition to being compressed by constricting scar tissue, are often distorted by the direct pressure of the gland itself.

Pathological Histology.

The most striking feature is the presence of dense fibrous tissue throughout the gland. In the early stages this is cellular, and in addition to a great increase in the number of fibroblasts, intermingled lymphocytes, plasma cells and histiocytes are seen. Later the connective tissue becomes less cellular and the intercellular substance is dense and hyaline (Figure I). Areas of lymphoid tissue may be present, but they are not so conspicuous as in lymphadenoid goitre. Relatively normal areas of thyreoid parenchyma may be observed surrounded by dense fibrous tissue. Indeed, some writers have considered that this one feature distinguishes the condition from lymphadenoid goitre. Areas of hyperplasia may be seen even when much of the gland has been destroyed. In these circumstances it may be compensatory, thus preventing the development of myxœdema. If the patient exhibits mild thyreotoxicosis, as some do, the hyperplastic epithelium is significant.

Areas of thyreoid cells which probably represent acini in process of destruction are present. They show pyknotic nuclei, vacuolated protoplasm and ill-defined cell outlines (Figure I). Masses of "colloid" without surrounding epithelial cells probably represent a later stage in the destruction of alveoli.

Areas of proliferating small acini may be seen associated with cellular connective tissue. This may easily be misinterpreted as being malignant, but mitotic figures are difficult to find, there are no great variations in the size and shape of the cells, and no invasion of blood vessels. The changes in the rest of the gland are more important in the exclusion of a carcinoma than is the morphology of these proliferative regions; for in the thyreoid, malignant disease gives a great variety of histological appearances. The correct interpretation of "frozen sections" in such cases as the above may be very difficult.

Rarely, one sees extensive metaplasia of the acinar epithelium. In such cases many of the vesicles are lined by a single layer of very flat cells; but in others the walls are composed of several layers, and transformations to typical stratified epithelium can be seen (Figure II). The occurrence of this type of epithelium has led to astonishing speculations as to its significance. One writer (Meeker), seeing a large vesicle cut obliquely as in Figure II, interpreted it as a remnant of the thyreoglossal duct. This led to a further suggestion that infection could have passed from the pharynx along this tract to the thyreoid and thereby have caused the disease. No actual duct could be found on dissection. In a case studied by the writer, almost every vesicle showed evidence of metaplasia, and all gradations were seen, from a single layer of flattened cells to stratified squamous epithelium. This occurrence was closely associated with the presence of periacinar lymphoid tissue. Many acini showing this change were devoid of stainable colloid, and in others it was reduced in quantity and fragmented.

Similar changes in the thyreoid have been produced experimentally in animals by the administration of diets deficient in both vitamin *A* and other essential foodstuffs. Yoshida claimed that he had produced it in guinea-pigs which received repeated injections of a toluene derivative. The writer repeated these experiments, but was not able to cause metaplasia. Possibly Yoshida's animals suffered from vitamin deficiency.

In addition to intravesicular giant cells, extravesicular giant cells of the foreign body type are often seen. Many are about 30μ in diameter, but some are considerably larger. Differences of opinion have been expressed as to their origin and significance. Some writers state that they arise from thyreoid epithelium, while others hold that they are developed from histiocytic cells. There is reason to believe that both forms occur, for in some the nuclei resemble tissue histiocytes and in others there is a close resemblance to thyreoid cells. Foreign body giant cells are found in pathological conditions surrounding material difficult of absorption, and they are probably of similar significance in the thyreoid gland.

The other form may result from the desquamation of thyreoid cells into the colloid. When the lining cells of the vesicle disappear the giant cell appearance results. The occurrence of foreign body giant cells has led to the diagnosis of tuberculosis in some instances, and Figure II shows how easily this mistake can be made. A careful search for tubercle bacilli shows them to be absent.

The condition of the blood vessels varies in different specimens. Pronounced thickening of the arterioles and small arteries is the rule, but in some instances these vessels appear to be normal. The number of capillaries is reduced.

Parathyreoid Glands in Riedel's Disease.

In the extensive literature reviewed the writer was unable to find any reference to the condition of the parathyreoid glands in Riedel's thyreoiditis. However, opportunity for study was afforded by one case in the present series. A parathyreoid gland, removed from a patient with Riedel's disease, proved to be histologically normal (Figure IV).

Clinical Features of Riedel's Thyreoiditis.

Riedel's thyreoiditis is a rare disease. Pemberton encountered it 48 times in 10,500 thyreoidectomies. Men and women are equally affected.

The majority of patients are in the fourth decade, but they may be younger or much older. The most conspicuous clinical features are referable to pressure from a large thyreoid gland and to constriction from surrounding fibrosis. Symptoms usually occur coincidently with the appearance of the goitre; but occasionally the cervical swelling may precede them by a period of months or years. Attention has been called to the fact that pressure symptoms are often out of all proportion to the size of the gland. The commonest are a dry cough and dyspnœa on exertion.

Unilateral thyreoid involvement is usually associated with much tracheal displacement and compression, and when both lobes are affected, there may be considerable narrowing and distortion. Although periglandular fibrosis is present in 80% of cases, involvement of the recurrent nerve is rare. Compression of the internal jugular veins may cause cyanosis and even œdema of the face and upper part of the neck. Dysphagia due to œsophageal compression is common.

While the patient often has a feeling of oppression in the neck, pain is conspicuously absent, although there is a little tenderness on palpation. Frequently the temperature is normal; usually there is no leucocytosis, and the cervical lymph glands are not enlarged.

One case studied by the writer was a striking exception to the general rule. The patient had a persistent otherwise unexplained rise in temperature, up to 100° F. at night, for several weeks prior to thyreoidectomy; this subsided promptly when the diseased gland was removed (Figure V).

In the early stages of the disease some patients show all the features of mild thyreotoxicosis. This has been denied by some writers, yet a study of their case histories often shows that the patients quoted suffered from palpitation, nervousness, tachycardia and loss of weight prior to reporting for treatment. More important diagnostically is the occasional presence of hypothyreoidism; this is almost unheard of in primary carcinoma of the thyreoid, so that its presence in a doubtful case would favour the diagnosis of Riedel's disease.

When both lobes and isthmus are involved the thyreoid is moderately enlarged and extremely hard; usually the surface is smooth, but it may feel more nodular clinically than at operation. The borders are difficult to define, and in typical cases thickening can often be felt extending into the subcutaneous tissues well beyond the confines of the gland. This is fixed to the surrounding structures, and when swallowing is attempted may either remain fixed or appear to undergo a curious rotatory movement. When one thyreoid lobe only is involved the hard swelling may be interpreted as a calcified nodule or a carcinoma. Usually, however, its outline is more difficult to define than most other nodules.

As a rule the symptoms are steadily progressive, and if untreated, the patient dies of asphyxia. In some instances myxœdema develops and the pressure symptoms regress. After the operative removal of only small portions of the gland, the swelling may diminish in size and the symptoms disappear. On the other hand, cases have been reported in which the removal of a large portion of the diseased tissue has been followed by a recurrence of symptoms.

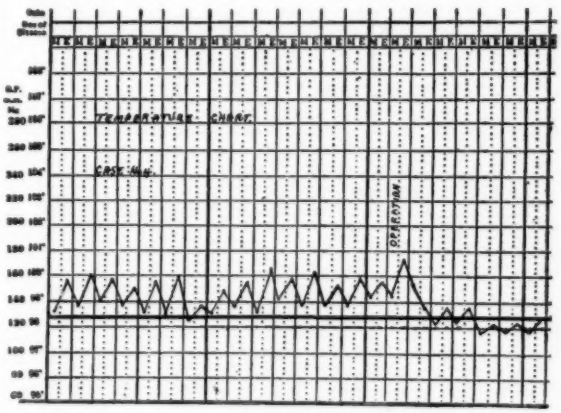

FIGURE V.
Temperature chart of a patient with Riedel's thyreoditis. It shows the rapid subsidence of the pyrexia after thyreoidectomy.

The Differential Diagnosis.

Helpful features in the differential diagnosis are the extreme hardness of the gland, its smooth surface and fixity, and the involvement of the subcutaneous tissues without pronounced constitutional disturbance or enlargement of the cervical lymph glands. If the features of myxœdema are present they favour Riedel's disease; but mild thyreotoxicosis may be a feature of both this and malignant disease. Some cases cannot be diagnosed correctly prior to operation. The most usual mistake is to diagnose a carcinoma.

Syphilis may be excluded by the absence of other signs of that disease and an absence of response to the Wassermann test. Tuberculosis should be diagnosed clinically only when there is evidence of other tuberculous foci, and pathologically only when the bacilli are cultivated from the gland.

"Adenomata" have been previously mentioned.

Unless extensive periglandular fibrosis is present, usually one cannot differentiate lymphadenoid goitre, although a rise in temperature favours the diagnosis of Riedel's disease.

Subacute infective thyreoiditis following such diseases as acute tonsillitis and influenza may be hard to distinguish; but the development of a painful and tender thyreoid swelling associated with infection of the upper portion of the respiratory tract is suggestive. No doubt some of the cases reported as of Riedel's disease are of this type, and indeed the thyreoid glands may be identical histologically.

Treatment.

Surgical Treatment.—The most satisfactory treatment is surgical removal of as much of the gland as is feasible without damage to the trachea or recurrent nerves. In other words, subtotal thyreoidectomy should be practised. Attempts to remove the whole gland are fraught with difficulties and may result in the patient's death. Division of the isthmus is a simple procedure and in some cases has been adequate to relieve compression; usually such treatment is unsatisfactory and at most gives temporarily relief. Removal of the isthmus and suture of the lateral lobes to the infrathyreoid muscles has been practised in order to relieve tracheal compression; but here again there is a great liability to the recurrence of pressure. Destruction of gland tissue by cautery is advocated by Sloan, but as the method lacks precision it has not received general support.

Deep X Ray and Radium Treatment.—Deep X ray and radium treatment have not been successful in Riedel's thyreoiditis, although patients suffering from lymphadenoid goitre, sometimes placed in this category, have experienced considerable improvement or "cure".

Lymphadenoid Goitre.

Lymphadenoid goitre may be classified as (*a*) diffuse lymphadenoid goitre and (*b*) focal lymphadenoid goitre.

Diffuse Lymphadenoid Goitre.

The synonyms of diffuse lymphadenoid goitre are: lymphadenoid goitre (Williamson and Pearse) (Joll); *struma lymphomatosa* (Hashimoto); chronic lymphoid thyreoiditis (Hashimoto's type) (Means); chronic lymphatic thyreoiditis (Hertzler).

As mentioned previously, the disease was described first by Hashimoto, in 1912, under the title *struma lymphomatosa*. Williamson and Pearse, in 1929, named it lymphadenoid goitre. Since 1928 numerous writers (Benson, and Shaw and Smith), following Ewing in not wishing to make any distinction between this and Riedel's thyreoiditis, have recorded many cases under the latter heading. In the writer's opinion the diseases should be considered separately, unless further evidence is obtained to show that they are different stages of the same disease. Cases in which lymphoid tissue is present as localized conspicuous nodules should also be separated from those in which the diffuse lesion is present. The former is best named "focal lymphadenoid goitre", the latter "diffuse lymphadenoid goitre". Such usage makes for accuracy in recording and does away with the practice adopted by some recent writers (for example, Vaux) of grouping together cases in which the focal lesion only is present with those of the diffuse disease. The belief expressed that the focal change is an early stage of the diffuse condition is at present unsupported by substantial evidence and is highly improbable.

Pathological Anatomy.—Both lobes of the thyreoid are as a rule uniformly enlarged and very firm; the size may be two or three times that of the normal gland. The surface is comparatively smooth and greyish-white in colour. Adhesions may be present between the gland and the capsule of pretracheal fascia, but not beyond this region. The cut section is often pale and greyish-white in colour; in other cases it is pale pink and mottled. The connective tissue septa, which are pinkish white in colour, can be seen outlining the pseudo-lobules.

Pathological Histology.—The essential feature of typical cases is the presence of abundant lymphoid tissue occupying a considerable portion of the enlarged gland. Much parenchyma is replaced by lymph nodules or diffuse lymphoid tissue. The thyreoid acini vary in size and appearance in different parts of the gland. The majority of vesicles have a diameter of 40μ to 60μ; these are frequently collapsed and star-shaped, being devoid of stainable colloid. Many acinar cells are seen in the lumina giving the appearance of giant cells. In other cases cell masses inside the vesicles show nuclei of several types, some resemble those of thyreoid epithelium, while others are evidently of histiocytic origin. Most of the cells lining the vesicles are between 9μ and 12μ in height and have granular protoplasm; the cell borders are frequently very indistinct, and fragmentation may be present. The histological appearance suggests that many of the acini are undergoing dissolution. In addition to the cell types described above, larger eosinophilic cells of an average diameter of 20μ are encountered frequently. These have relatively small pyknotic nuclei and abundant granular protoplasm (Figure VI). Sometimes these are present as occasional cells in a vesicle where the remaining cells are of the usual type, or the acini may be entirely composed of these large cells, while in separate regions small groups of them may be seen irregularly scattered in the lymphoid or fibrous tissue. These eosinophilic cells are similar to or identical with the so-called "parafollicular" cells, first described by Baber and later by Hürthle. They are not confined to this disease, although they are almost invariably present in some parts of the gland. They are sometimes seen in otherwise normal thyreoid glands, toxic goitres and tumours. There is considerable diversity of opinion as to their nature. Some consider them to be parathyreoid rests (Wilensky and Kaufman). Their widespread distribution in the thyreoid and their presence in vesicles together with other more usual types of thyreoid cell make such a view untenable. Clute, Eckerson and Warren regard them as degenerating cells, while others consider that they are in an active state of proliferation. Hellwig compares them with eosinophilic cells sometimes seen in the ducts of the breast in chronic mastitis, and regards them as having been produced by metaplasia due to some irritant. The present study shows that they may be found in areas of active proliferation or degeneration, and in some cases in which neither process is in evidence. Their appearance in such diverse conditions suggests that they result from some nutritional alteration in the cellular environment, the exact nature of which is unknown. This alteration in cell morphology is probably associated with a change in metabolic activity. While some writers state that normal vesicles are not found in the disease, microscopic examination of multiple sections invariably reveals their presence. Areas of active papillary hyperplasia are often seen, and can be correlated with the signs and symptoms of thyreotoxicosis when they are present. Colloid storage is evident in some portions, and indeed one may see almost any variety of histological structure found in other abnormal states. Lymphoid tissue is seen between the vesicles and even invades them. Lymph nodules, in spite of statements to the contrary, often have clear "germinal" centres. Between these the lymphocytes, plasma, and reticuloendothelial cells are arranged diffusely. This pronounced hyperplasia has led to the erroneous diagnosis of lymphosarcoma or anaplastic carcinoma. Fibrosis of variable degree is common and hyalinization of the connective tissue may be a prominent feature. Ewing and others consider that as the disease progresses the lymphoid tissue is replaced by fibrous tissue; but there is no proof of this. Indeed, in those few instances in which biopsy on a gland has been performed at intervals of several years, the transformation has not been in evidence. Deductions therefore have been drawn from analogy with other diseases.

The thyreoid gland is usually less vascular than normal. In some specimens the capillaries are greatly reduced in number, and the arterioles show endarteritis, but often these are normal.

The Pathogenesis of Lymphadenoid Goitre.—All views on the pathogenesis of this disease are largely speculative; the individual theories will be considered in turn. (*a*) Lymphadenoid goitre is a local manifestation of a constitutional disorder. This non-committal theory which is widely held explains little, for one is entirely ignorant of the nature of the "constitutional disorder". (*b*) The disease results from diets deficient in vitamin *A* and other essential foodstuffs. While similar histological changes can be produced in the thyreoid of rats by the administration of diets deficient in vitamin *A* and other substances, similar deficiencies cannot be found in the diet of patients suffering from lymphadenoid goitre. It is improbable, therefore, that an inadequate diet plays an essential part

in the human condition. (*c*) Hellwig considers that lymphadenoid goitre results from an increased output of thyreotropic hormone which "releases" colloid from the vesicles and so interferes with their function. The commonly found star-shaped acini are thought to result from that process. Were the hypothesis correct, one would expect the colloid "release" to be associated with severe thyreotoxicosis, and this is a most unusual finding. (*d*) Some writers regard lymphadenoid goitre as being a chronic inflammatory disease, an early stage in the development of Riedel's disease; but organisms have seldom been found in the gland and the histological evidence for the inflammatory nature is inconclusive. The present state of knowledge is such that any committal statement cannot be made either on the nature of the disease or on its ætiology.

Clinical Features.—Nearly all the reported cases are in women of about fifty years of age. The onset is usually insidious, both the enlargement of the gland and the pressure symptoms occurring as a rule about the same time. Cough, dysphagia and shortness of breath on exertion are frequent, while in the early stages evidence of mild thyreotoxicosis is noticed occasionally. Nervousness, loss of weight and strength, palpitation, and even exophthalmos may be presenting symptoms. Later in the course of the disease subthyreoidism may be noticeable. The thyreoid gland is enlarged and hard, the surface being smooth; both lobes are uniformly affected, although one may be larger than the other. It may also be slightly tender to pressure. Adhesions to surrounding structures are absent, and the movement of swallowing is not affected. Because of the uniform involvement of the whole gland, the smooth surface, and the absence of fixity to surrounding structures, the surgeon usually can distinguish the lesion from carcinoma. The presence of any features of myxœdema following those of mild thyreotoxicosis is particularly significant. In many instances Riedel's disease cannot be distinguished from lymphadenoid goitre on clinical grounds. The difference in age and sex incidence, and the absence of periglandular fibrosis in diffuse lymphadenoid goitre, are sometimes helpful features. The effect of deep X ray treatment in rapidly diminishing the size of the gland and alleviating the symptoms has recently been suggested as an important diagnostic as well as a therapeutic measure (Renton, Charteris and Heggie).

The Treatment of Lymphadenoid Goitre.—Surgical treatment up to the present time has proved most efficacious, the operation of choice being subtotal thyreoidectomy. This makes possible both an accurate diagnosis and a removal of most of the diseased tissue. The drawback of the method is that in any individual case one cannot be certain how much of the gland should be left behind. A recurrence of pressure symptoms follows the removal of too little, while myxœdema results from the removal of too much. Indeed, nearly 40% of patients develop post-operative hypothyreoidism. This cannot be attributed wholly to the treatment, for spontaneous myxœdema is not an infrequent accompaniment of the disease. In view of this the patients should be kept under close observation for several years after they have received treatment.

Recently a series of six patients treated by radium has been reported to show loss of symptoms over a period of from two to four years without the development of myxœdema. The authors consider that with the dosage used, the diminution in the size of the glands is sufficiently pronounced to be of diagnostic significance. Means previously reported a patient successfully treated by similar dosage of deep X rays (Renton, Charteris and Heggie). However, the results of this small series cannot be considered to have proved surgical treatment inferior, particularly as without biopsy the surgeon is not certain of the actual condition he is treating.

Focal Lymphadenoid Goitre.

The writer has found extensive areas of lymphoid tissue in 3% of toxic nodular goitres. When such are sufficiently conspicuous and localized to warrant attention, the condition may be spoken of as focal lymphadenoid goitre.

Pathological Anatomy.—The thyreoid may show all those features present in both "toxic" and "non-toxic" glands in addition to the focal lymphadenoid areas. The nodules appear as greyish white, whitish, or yellowish opaque regions in the thyreoid substance; they are not encapsulated and gradually merge into the surrounding parenchyma. This tissue is firm to hard in consistency. In one case the firm, whitish areas seen at operation led to the erroneous diagnosis of carcinoma; but their distribution in both lobes without penetration of the capsule should help in the differentiation from malignant disease. Usually at operation it is impossible to differentiate the condition from the more usual types of nodular goitre. When examined on section the whitish areas are of firmer consistence than those resulting from localized areas of thyreoid hyperplasia, and therefore can be distinguished from them.

Pathological Histology.—When sections are stained with hæmatoxylin and eosin the blue lymphoid areas are very conspicuous. They may stand out sharply in contrast with the rest of the section as in Figure VI, or gradually merge into the adjacent parenchyma. The surrounding tissue may be hyperplastic, or be the site of colloid storage. The lymphoid areas show changes identical with those seen in diffuse lymphadenoid goitre.

The Relationship Between Diffuse Lymphadenoid Goitre and Focal Lymphadenoid Goitre.

A study of nodular "toxic" goitres shows the presence of large focal areas of lymphoid tissue in at least 3%. The histological changes seen in these areas are identical with those in Hashimoto's thyreoiditis. Vaux suggests that the focal lymphadenoid areas represent an early stage of the diffuse condition; but since many patients with lymphadenoid goitre give no evidence of having passed through a "toxic" nodular phase, this cannot be accepted. Moreover, if this were the case one would expect many patients with "toxic" goitre to develop myxœdema while at the same time retaining a large, hard, thyreoid gland. The histological picture suggests that coincidently with the appearance of the lymphoid tissue there occur collapse of thyreoid vesicles, desquamation of many of the parenchymatous cells, and loss of colloid. In many instances vesicles disappear completely. Whether or not this is a result of "exhaustion" due to over-stimulation with thyreotropic hormone, as suggested by Hellwig, or to some impairment of the blood supply to the tissue, is uncertain. Possibly the breakdown of thyreoid tissue may result in the appearance of lymphocytes and reticulo-endothelial cells, but more probably both changes result from a common cause. At the present time there is no good reason for classifying typical hyperplastic thyreoid glands, with localized macroscopic nodules of lymphoid tissue, as exhibiting Riedel's thyreoiditis or diffuse lymphadenoid goitre.

The Relationship Between Diffuse Lymphadenoid Goitre and Riedel's Thyreoiditis.

Since there are appreciable differences between typical cases of Riedel's disease and those of diffuse lymphadenoid goitre, it is expedient to separate them as distinct entities, so that with accumulated knowledge more just comparison may be made and the relationship determined. The policy of reporting series of cases of either condition under the term "Riedel's disease" has led to confusion rather than to clarity, and many authors have fallen into error by drawing conclusions from three or four cases without giving due consideration to all that have been reported.

Less than 150 cases of Hashimoto's syndrome have been sufficiently well described in the literature to allow one to make a diagnosis of lymphadenoid goitre. Over 95% of these patients were women, the majority being about fifty years of age. The average age incidence is therefore slightly higher than for Riedel's thyreoiditis. This does not support the idea that diffuse lymphadenoid goitre is an early stage of Riedel's disease, in which the average age incidence is about forty years. A study of the sex incidence shows that Riedel's thyreoiditis occurs with almost equal frequency in men and women, again contrasting with lymphadenoid goitre. In 30% of cases

of Riedel's disease only one-half of the thyreoid gland is involved, while in diffuse lymphadenoid goitre both lobes are invariably affected. When pieces of tissue have been removed from a lymphadenoid gland at intervals of between one and a half and two and a half years, no transition from the lymphoid to the fibrous phase has been observed. Some patients suffering from Riedel's thyreoiditis have extensive fibrosis in the cervical muscles, the connective tissue outside the gland causing pronounced constriction of the trachea, the œsophagus and the great vessels of the neck. Occasionally the overlying skin is ulcerated. In these circumstances one could not regard the disease as being a metabolic disorder of the thyreoid gland, a plausible theory held by many with regard to Hashimoto's disease. The possibility of an infective agent causing Riedel's disease is most likely. In lymphadenoid goitre the lymphoid tissue is confined to the gland and the cervical structures are not involved. Other points of distinction may be mentioned and analysed. The duration of symptoms is usually longer in lymphadenoid goitre, recovery is slower after operation, and the incidence of post-operative myxœdema is higher.

One reason for this is that in Riedel's thyreoiditis subtotal thyreoidectomy is often impossible technically, and the patient is therefore left with a considerable amount of thyreoid tissue. On the other hand, there is no special difficulty in the removal of a lymphadenoid goitre, and a generous subtotal thyreoidectomy is the rule. This is not a complete explanation, for a greater proportion of patients with lymphadenoid goitre show hypothyreoidism prior to operation. Little can be deduced from the average duration of symptoms in either condition, for in individual cases the times vary considerably, and the number of cases recorded is too small to warrant statistical evaluation. The chief histological distinction is that on the one hand the thyreoid is composed largely of lymphoid tissue, and on the other a considerable portion of the parenchyma is replaced by dense fibrous tissue.

In various infective processes in the body lymphoid tissue may be found in abundance in one stage of a disease and be replaced later by fibrous tissue; but the significance of the lymphoid tissue in the thyreoid is unknown. Also, there is no direct evidence that in this situation it is extensively replaced by fibrous tissue to give the typical histological appearance of Riedel's thyreoiditis. However, the possibility of a relationship between the two cannot be excluded while the ætiological factors concerned in both diseases are obscure.

The Relationship of Diffuse Lymphadenoid Goitre to Myxœdema.

Some writers consider that lymphadenoid goitre is an early stage in the development of "idiopathic" myxœdema (Williamson and Pearse). While it is true that a number of patients suffering from diffuse lymphadenoid goitre develop myxœdema even prior to operative treatment, the history of the vast majority of patients with that condition does not indicate that it follows an enlarged thyreoid gland. Indeed, the pathological processes appear to be essentially dissimilar. For some unknown reason the thyreoid parenchyma atrophies in myxœdema, and a replacement fibrosis results.

In lymphadenoid goitre there is evidence of an active proliferation of lymphoid tissue, which causes a considerable increase in the size of the gland. We have no reason to believe that this is ever replaced by large amounts of fibrous tissue.

Acknowledgement.

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ILLUSTRATIONS TO THE ARTICLE BY Dr. NORMAN M. HARRY.

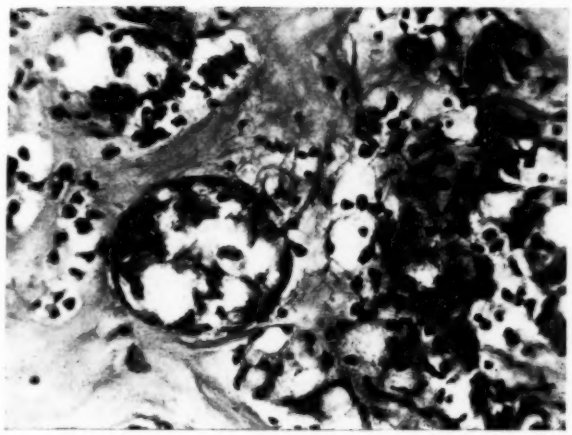

FIGURE I.
Riedel's thyreoiditis, showing disintegrating acini in dense fibrous tissue. × 320.

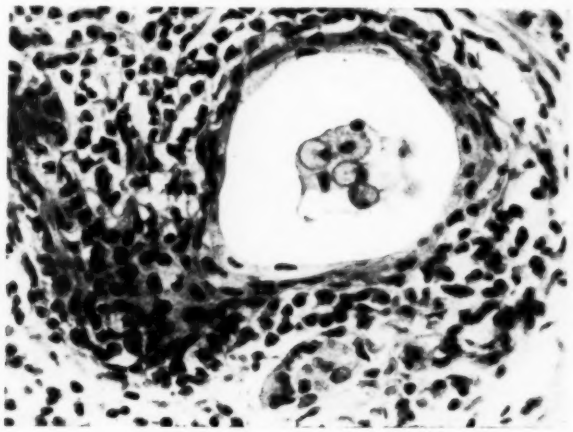

FIGURE II.
Riedel's thyreoiditis, showing stratified squamous cell metaplasia. × 320.

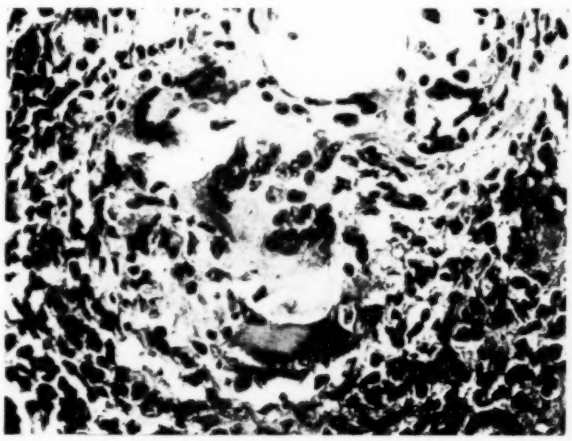

FIGURE III.
Riedel's thyreoiditis; a group of foreign body giant cells is seen. × 300.

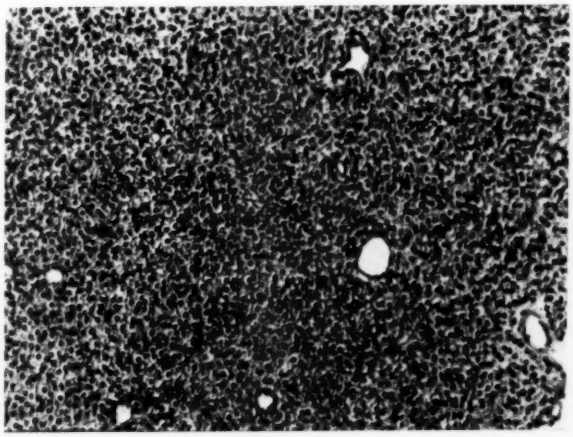

FIGURE IV.
Low power photomicrograph of a normal parathyreoid gland removed from a patient with Riedel's thyreoiditis.

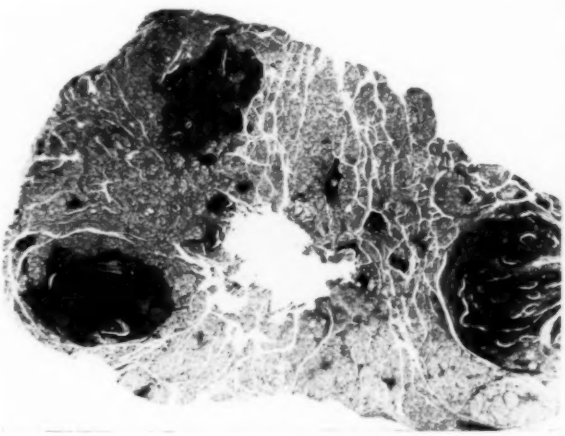

FIGURE VI.
Focal lymphadenoid goitre; photomicrograph of a section stained with hæmatoxylin and eosin showing lymph nodules. × 4.

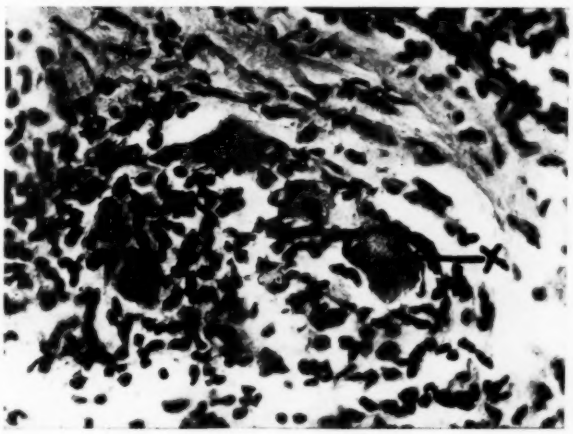

FIGURE VII.
Photomicrograph showing a group of Hürthle cells (×) in a lymphoid area.

ILLUSTRATIONS TO THE ARTICLE BY DR. P. L. HIPSLEY.

FIGURE I.

Pyelogram showing slight dilatation of the renal pelvis on the right side, due probably to a temporary block in the ureter by a calculus which had passed spontaneously. The child had had an attack of colic for which his appendix had been removed. The left kidney is not functioning, but its shadow is much enlarged owing to the small calculus (see arrow) in the lower end of the left ureter.

FIGURE II.

Pyelogram after the stone in the left ureter had passed spontaneously; both kidneys are functioning. This case is exactly similar to all the cases reported in the series, excepting that the blockage in both ureters did not occur simultaneously.

ILLUSTRATIONS TO THE ARTICLE BY DR. DOUGLAS ANDERSON.

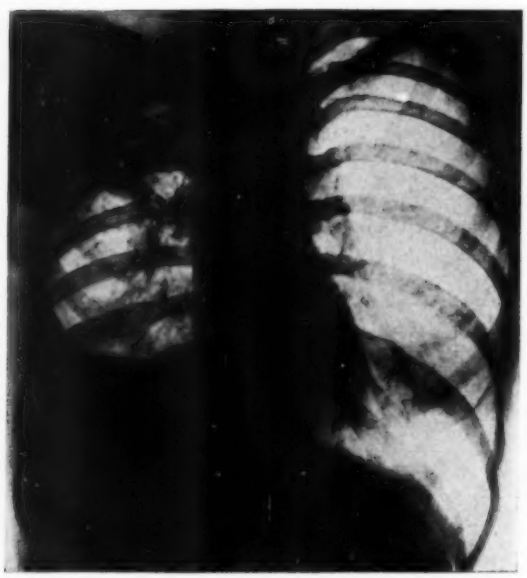

FIGURE I.

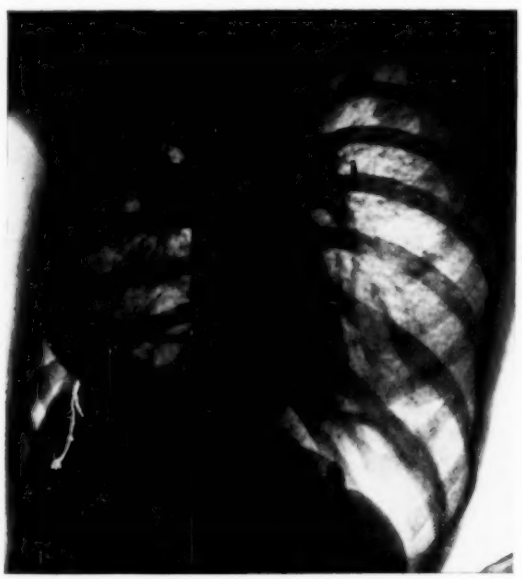

FIGURE II.

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A CRITICAL REVIEW OF CASES OF SO-CALLED REFLEX ANURIA, WITH THE REPORT OF A CASE.

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FROM time to time in our medical journals cases of anuria in children have been reported, and the impression gathered from the reports is that for some unexplained reason the kidneys suddenly fail to function, and that there is a cessation of renal excretion, the kidney pelves being left empty of urine. As this condition usually follows some surgical procedure, it is often referred to as surgical anuria, and as it is presumed to be in some way due to the operation, possibly to some disturbance of the reflex nervous mechanism of the kidney, it is sometimes called reflex anuria.

Various types of operation have preceded the anuria. Sometimes the passage of a ureteral or urethral catheter appears to be the exciting cause. In the series of cases quoted in this paper, in five (Cases I to V) anuria followed appendicectomy, in one (Case VIII) it followed nephropexy, in another (Case IX) it followed the setting of a fracture, and in three (Cases VI, VII and X) no operation had preceded the onset of anuria.

As the cause of the anuria is always obscure, the treatment adopted is usually empirical. Several widely different methods have been employed with equally satisfactory results. Some of the methods used are as follows: pyelostomy or nephrostomy on one side only (Case I); nephrostomy on both sides (Case V); ureteral catherization, either alone or in conjunction with spinal anæsthesia (Case VIII); decapsulation of the kidneys alone (Case IX), or in conjunction with nephrostomy;[1] intravenous injections of hypertonic saline solution, and solutions of glucose or of sodium sulphate (Case III). Cases have also been reported in which recovery followed diathermy to the loins, and in some recovery has taken place when no special treatment has been instituted. In the cases which I have in mind the condition was very similar in its mode of onset and in its symptomalogy; but the methods of treatment employed appear at first sight to be very dissimilar. If a satisfactory explanation could be given of the mode of action of the various methods of treatment which have been successfully used, then one would be in a position to formulate some hypothesis as to the cause. I propose to try to demonstrate that the condition is almost certainly due to calculi lodged in both ureters, or in other words, that the lesion in all cases is the well-known condition, calculous anuria, but radiography and other methods of urological investigation have failed to reveal a calculus. In support of this hypothesis is the fact that all the methods of treatment mentioned above might be successful in removing an obstruction if it were due to a small stone in the ureter, and I can think of no other condition in which such different methods of treatment would be effective. The symptoms recorded in all cases are similar to those commonly seen in renal colic due to stone. There has been in the great majority of cases severe pain in the loins, generally associated with vomiting. In most cases (Cases I, II, III, IV, V, VII and IX) hæmaturia was present. Pyrexia has usually been absent, or at most there has been a very slight rise in temperature. There has been no evidence of pyelitis beforehand, although it has frequently been present afterwards; and no evidence of kidney damage has been found either before or after the attacks. In some cases the X-ray film has failed to reveal a calculus; but in some a shadow suggestive of a calculus has been seen, and in one of the cases reported above (Case X) a small calculus was passed after the attack.

Most of the above cases were in children, and we know that renal calculi are not uncommon in children. Garrod, Batten, Thursfield and Paterson[1] quote Thomas and Tanner, of the United States of America, who, after reviewing a series of 203 cases, found that the average age of children with renal calculus was seven years.

Sheldon[2] states that "during the first few weeks of life large numbers of uric acid crystals are passed in the urine", and he also states that "post-mortem examinations on infants often show uric acid concretions no larger than a split pea in the renal pelvis". On examining the records of the Royal Alexandra Hospital for Children, Sydney, I found that there had been 30 cases of undoubted renal calculus at the hospital in the eleven-year period between October, 1927, and September, 1938, and in all of these cases the X-ray film revealed the calculus. The age distribution of these cases was as follows: twelve months, one; two years, three; three years, three; four years, one; five years, two; six years, six; seven years, five; eight years, one; nine years, three; ten years, two; eleven years, one; twelve years, one; fourteen years, one. These figures, however, do not include all the patients with renal calculi admitted to the hospital; there must have been many patients in hospital with renal calculi whose condition was not recorded as such, because of an absence of X-ray evidence. It is not always possible, even in adults, to demonstrate a stone by means of radiography alone, and in children with a small uric acid calculus it would be almost impossible.

One one occasion I removed three uric acid calculi, each as large as a pea, from the pelvis of a child's kidney; no shadow was to be seen in the skiagram.

It is interesting to note that several patients with renal calculus have had the appendix removed, and the children have returned later with a right-sided calculus. I can find no record in our own journals of any case reported as calculous anuria in a child. Since renal calculi are not infrequent in children, cases of calculous anuria must occur sometimes, and I would suggest that their frequency would be about that of the cases reported as reflex anuria. Calculous anuria from its very nature must be a rare occurrence. Cases in which a calculus completely blocks one ureter obviously occur more frequently than cases in which a bilateral ureteral block occurs; but these cases do not attract the same attention, because the kidney on the unobstructed side continues to function, and so there is no danger of uræmia. The following is the report of such a case (see Figures I and II).

G.B., aged ten years, was admitted to the Royal Alexandra Hospital for Children on March 20, 1940, because of severe pain in the left loin. The urine contained blood visible macroscopically. He had had a similar attack of pain on the right side some time before, and the appendix had been removed. An excretion pyelogram showed that the right kidney functioned normally, but that there was a mild degree of hydronephrosis on this side. The left kidney excreted no dye whatever, but its shadow, which could be easily seen, was greatly enlarged. The radiographer reported that there was a tiny calculus in the lower end of the left ureter. A few days later a cystoscopic examination was made and an attempt was made to catheterize the ureters. There appeared to be a complete block on both sides at a point about one inch from the uretero-vesical junction. A second excretion pyelogram made shortly before the cystoscopic examination showed both kidneys functioning normally, and also revealed that there was some slight degree of hydronephrosis on the left side as well as on the right.

In spite of the complete block of the ureter on one side, there was no reflex anuria in this case. There is no doubt that there had been in this case a calculus on the right as well as on the left side; the dilatation of the renal

[1] In one case which came under my notice a successful result followed decapsulation followed by manual compression or squeezing of both kidneys.

pelvis was evidence of this, but calculous anuria did not occur because both sides did not become blocked simultaneously. I have no doubt that if the left kidney had been explored after the first pyelogram was taken, the pelvis would have presented features similar to those in other cases which have been reported as reflex anuria; that is to say, the pelvis would have been found to be tensely distended with urine. No special treatment was given and the obstruction disappeared spontaneously. As has been the case in most of the patients with so-called reflex anuria, the stone was not recovered in spite of the fact that one was demonstrated radiologically.

The object of the treatment used in the cases recorded in this paper (Cases I to VIII) has been to try to stimulate the kidney to resume its lost function, and not to displace a calculus. It is not realized that in these cases there is acute retention in the renal pelvis, which is tensely distended with urine owing to a block in the ureter (see Cases IV, V, VI and VII), and the kidney ceases to function only because of this block. The different methods of treatment used would be more likely to bring about the displacement of a calculus which had lodged in the ureter, than to result in the reestablishment of filtration of urine when this function was in abeyance. One fails to see how decapsulation of the kidneys or pyelostomy or nephrostomy could possibly relieve these patients, unless the kidney pelvis was distended with urine beforehand. The mere opening of an empty kidney pelvis would be futile.

Before passing on to the report of a fourth case of the kind, which has come under my own observation, I should like to quote Samson Wright[3] to show what happens when a ureter is suddenly and completely blocked:

> If the ureter is ligated, filtration of urine ceases when the pressure in the ureter reaches 75 mm. of mercury. If the plasma is greatly diluted by the injection of saline, and the protein osmotic pressure reduced, filtration goes on till a higher ureteric pressure is attained *e.g.* 90 mm. of mercury.

I believe that these observations have an important bearing on the clinical course of this condition, and explain why the various and apparently dissimilar methods of treatment are frequently successful.

Report of a Case of So-Called Anuria in a Young Child.

W.W., a male patient, aged one year and ten months, was in the Royal Alexandra Hospital for Children, Sydney, under the care of Dr. Donald Vickery, who referred him to me. The following history was obtained. On July 7 the child was peevish and fretful, refused all food, and had mild diarrhœa and a normal temperature. He remained in much the same condition for three days, and then had a screaming attack, due apparently to severe abdominal pain. There was no abdominal rigidity. His temperature at this stage was 99·6° F. and the pulse rate was 110 per minute. Four days later the mother noticed that no urine had been passed for twenty-four hours, and she was not sure whether any had been passed for some considerable time before this. A few drops of dark blood-stained urine were obtained by catheterization. I saw the child first on July 9. He was pale, had a temperature of 99° F., a pulse rate of 116 per minute, and was tender over both loins, particularly on the left side. He had had complete suppression of urine for twenty-four hours and possibly longer. It was decided to perform a nephrostomy on one side and follow with a similar operation on the other side only if the kidney on this side failed to function after the first operation. Although I realized that it did not matter which side was selected in these cases for the first operation, when it was decided to operate on one side only at first, I chose the left, as the pain was more severe on this side.

Under ether anæsthesia the left kidney was exposed, and, as was expected, the left renal pelvis was found to be tensely distended. A small puncture wound was made in the pelvis and the urine spurted out under considerable pressure. A soft rubber catheter was then passed, first through the incision in the pelvis and then through the cortex of the kidney and out through the loin. The end of the catheter was left just inside the pelvis, and the small incision in the pelvis was closed with fine catgut. Before the pelvis was closed, however, an attempt was made to pass a ureteral catheter along the ureter from the pelvis; but a complete block was encountered at a point about three inches from the uretero-pelvic junction. Twenty-four hours later no urine had been excreted from either kidney. A solution of sodium iodide was injected along the drainage catheter; this, after X-ray examination, was found to be kinked in the depths of the wound, and this probably explained why the kidney failed to function. The X-ray film also showed a small shadow in the ureter, which the radiographer thought was possibly due to a small calculus. It was then decided to perform a nephrostomy on the other side, and at the same time to adjust the tube in the side already operated on. After the second operation both kidneys began to function immediately and the child made an uneventful recovery.

Comment.

This is the fourth case which I have reported. Two cases[4] (Cases V and VI) were reported in THE MEDICAL JOURNAL OF AUSTRALIA of December 2, 1933, at page 758, and one[5] (Case IV) was reported in THE MEDICAL JOURNAL OF AUSTRALIA of January 4, 1936, at page 19. All these cases have been dealt with in a somewhat similar manner to the one here reported; all the patients have recovered, and have remained well ever since operation, with the exception of the child in Case IV, whom I saw again recently because of a severe attack of renal colic. A complete urological investigation made on this patient five years after the original attack revealed numerous oxalate crystals in the urine; this probably accounted for the second attack of renal colic.

Two of the cases (Cases IV and V) occurred after appendicectomy; but the other two (Cases VI and VII) did not follow any operation. In the cases following appendicectomy the appendix did not show evidence of gross damage, although at the time the appendix was thought to be sufficiently inflamed to be the cause of the original pain.

With regard to the treatment adopted in these cases, in one case both decapsulation and nephrostomy were performed on both sides, and both kidneys immediately began to function; in this case both kidney pelves were distended. In another case nephrostomy was performed first on one side, and this kidney began to function immediately; but the other kidney did not function until a nephrostomy was performed on it also a few days later. Both kidney pelves were found at operation to be distended. In the third case nephrostomy, but not decapsulation, was performed on one side only, and the pelvis was distended with urine on this side. The other kidney was not operated on, yet both kidneys commenced to excrete urine after the operation. In the fourth case herein reported, the kidney pelvis was found to be distended with urine on both sides, and a nephrostomy was performed on both sides, at different times.

A case reported by S. P. Barnett and F. Schlink[6] also demonstrates the futility of decapsulation and the value of nephrostomy in the treatment of these cases. Their patient was a girl, aged thirteen years (Case II), and decapsulation was performed on one side and decapsulation and nephrostomy was performed on the other. The kidney treated by the double operation began to function within one hour of the operation, and the kidney treated by decapsulation alone began to function after three days.

A case is reported by Hyman[7] (Case IX) in which recovery followed an operation described as decapsulation; but in this case a section of the renal cortex was removed from both sides for purposes of histological examination, and urine drained from both lumbar incisions within twenty-four hours of the operation. The urine must have found its way through the portion of the cortex from which the section had been removed; hence the operation was really nephrostomy and not decapsulation as stated.

There is a case often quoted by Sydney surgeons which was supposed to demonstrate the value of decapsulation. The surgeon exposed the kidneys and decapsulated them without bringing them on to the loin, so that it would have been impossible for him to see the state of the pelves. It is said that at the time of the operation both kidneys were firmly compressed, a procedure which might easily have the effect of displacing a small calculus in the ureter.

Cases have also been reported in which intravenous injections alone have been followed by recovery. A case of this kind is reported by J. G. Whitaker and S.

Williams,[9] and I shall refer to this method of treatment later.

In another case reported by Basil-Jones[9] a cure was effected by pyelostomy performed on one side only.

Cubitt[10] reports a case in which an adult was cured by ureteral catheterization and intravenous injection of saline solution.

Anuria Following the Administration of Sulphapyridine.

Recently, cases of anuria following the administration of sulphapyridine have been reported by Carroll, Shea and Pike,[11] and also by Backhouse.[12] It has been demonstrated that in these cases crystals of an acetylated compound of the drug form in the urine, and may become blocked in the ureter in the form of concretions or small stones. Backhouse[12] reports a series of cases of hæmaturia and anuria of short duration, in which the symptoms described were identical with those of renal calculi. The pain in these cases was so severe that morphine had to be administered to prevent the patients from crying out. Hæmaturia was present in all cases. In the last case reported by Backhouse[12] the anuria lasted for eight days, and suddenly disappeared after repeated intravenous injections of glucose and saline solution. Backhouse is of the opinion that the anuria in his last case was largely a reflex phenomenon.

It would appear to me that the anuria in all these cases was undoubtedly due to a complete bilateral ureteral block with small calculi; and the only reason that we do not see more cases of anuria following the administration of sulphapyridine is that the chances of a simultaneous bilateral ureteral block must necessarily be small. No doubt unilateral and so unrecognized cases probably occur more frequently. Seeing that the use of sulphapyridine is increasing rapidly, I have no doubt that more and more of these cases will continue to be reported.

The Theory of Reflex Anuria.

If it can be proved that the term "reflex anuria" is a misnomer, then obviously it is unnecessary to use it in order to explain the occurrence of suppression of urine in the cases under consideration. I believe that the so-called anuria in Cases I to X reported in this paper can be satisfactorily explained by another hypothesis—namely, that it was due in all cases to bilateral calculi; or in other words, that they were all cases of calculous anuria.

There are, according to Joly,[14] three types of anuria: (i) pre-renal, due to a fall in blood pressure below 70 to 90 millimetres of mercury, when renal excretion ceases; (ii) renal anuria, due to changes in the renal epithelium, as in chronic renal disease; (iii) post-renal or obstructive anuria, which must be bilateral or else must involve the only functioning kidney. In a few cases one kidney is obstructed and the other temporarily thrown out of action either by reflex inhibition or by an excessive fall of blood pressure. Joly qualifies this, however, by stating that "in every case there is a cessation of renal excretion, it is never simply a retention of urine in the renal pelvis".

In my own series, Cases IV to VII, I had an opportunity of examining seven kidneys of the four patients, and in each case there was definitely acute retention of urine in the renal pelvis; hence they cannot be classed as reflex anuria, but as post-renal or obstructive anuria, and it is only necessary to define the type of obstruction. There are two possible types of obstruction to be considered: (i) spasm or achalasia of a section of both ureters; (ii) obstruction due to a calculus alone or with some stricture of the ureter, congenital or otherwise.

Spasm and achalasia can be ruled out as a cause of the obstruction, because such a condition would be hardly likely to commence suddenly and after lasting a week or more as suddenly to disappear, never to return again. Those who support the theory of achalasia may draw a parallel with such a condition as the well-recognized achalasia of the cardiac sphincter of the œsophagus, which persists for years or until cured by dilatation. But the sequence of events in the cases under consideration is entirely different and would be the natural sequence if the block was due to a calculus. It would be possible for pre-renal anuria of a very temporary nature to occur when shock associated with a low blood pressure was present; but the patients under consideration were not suffering from prolonged shock, and they definitely were not suffering from pre-renal anuria. Joly himself states that "the theory of reflex anuria is not proved and the bulk of evidence is against it".

All attempts at producing reflex anuria experimentally have failed. Joly quotes one authority as having ligated one ureter in each of a series of 100 dogs without the production of a single case of suppression of urine. The ureter is frequently damaged, cut or ligated accidentally during the course of pelvic operations; but the other kidney invariably continues to function when it has been normal prior to the operation. Numerous cases occur of complete blockage of one ureter due to calculus, similar to the case of G.B. reported in this paper; but the other kidney continues to function.

Quimby[15] has demonstrated that a transplanted kidney will function and will go on functioning indefinitely. Such a kidney has no possible connexion with the central nervous system. Hence there is no evidence that the so-called reflex anuria has any existence in fact, and the term should be abandoned.

The Relationship between Anuria and Surgical Operations.

Most of the cases of anuria reported in THE MEDICAL JOURNAL OF AUSTRALIA (Cases I to V) have followed appendicectomy, and in most of these cases one cannot be sure that the colic prompting the operation was not renal rather than appendicular. In none of the cases was the appendix frankly gangrenous or perforated. Moreover, if the operation of appendicectomy was sometimes a direct cause of anuria, then, since this operation is probably performed more frequently than any other, one would expect anuria to be a very common occurrence, whereas it is exceedingly rare. It is rather significant that in six out of the ten cases summarized in this paper hæmaturia and renal colic were present. Although an operation such as appendicectomy cannot be the direct cause of anuria, the fact that the anuria occurs soon after the operation suggests a possible connexion between the operation and the onset of anuria. In cases in which a renal calculus happens to be present during the course of some surgical procedure, the recumbency enforced by the operation and the higher concentration of urine incidental to the lessened fluid intake before and after operation may lead to an increased deposition of uric acid, oxalates or other crystals in the urine within the kidney pelvis, and so may possibly add to the size of a stone, or may even cause the formation of a small stone when one was not present before operation. Such a stone would tend to be forced into the ureter at about the time when the patient first sat up or moved about—that is, about the fifth to the tenth day, when most of the patients show the first sign of colic. J. Leon Jona[16] shows how sensitive the kidney pelvis is to movement. He has recorded experiments showing that in the recumbent position the pressure within the renal pelvis is at its lowest, but in the sitting position the pressure is immediately increased.

We know also that calculi sometimes form in the kidney during long periods of recumbency, as in patients confined to bed for long periods with tuberculous disease of bone, chronic osteomyelitis or infantile paralysis. I had a patient recently suffering from tuberculous disease of the hip-joint who developed a renal calculus, and Webster[17] has written on this subject.

Anuria is sometimes reported as following the passage of a ureteral catheter. I believe that in these cases, as the investigation is obviously being carried out for the elucidation of some condition which has been causing symptoms referable to the urinary tract, the occurrence of the anuria is possibly evidence that the patient has bilateral calculi which had not been demonstrated radiologically. The case reported by Hyman (Case IX) occurred after fractures of the femora had been set. In this case hæmaturia was noted; this was evidence of a stone being present.

TABLE I.

Summary of Ten Cases of So-called Reflex Anuria.

Number.	Sex of Patient.	Age. (Years.)	Operation (if any).	Time of Onset.	Signs and Symptoms.	Treatment.	Result.
I	M.	9	Appendicectomy.	5th day.	Tenderness in both loins. Hæmaturia. Both ureteric orifices red and œdematous. Attempt to catheterize ureters failed.	Pyelostomy of left kidney on ninth day. Glucose solution (5%) intramuscularly seventh day; 300 cubic centimetres of 4·28% sodium sulphate solution intravenously on eighth day.	Recovery. Left kidney functioned soon after pyelostomy. Right kidney began to function six days later.
II	F.	13	Appendicectomy.	8th day.	Pain in lumbar regions. Hæmaturia. Ureteral catheter met with obstruction two inches from vesical orifice on both sides.	170 cubic centimetres of isotonic sodium sulphate solution intravenously. Spinal anæsthesia. Decapsulation of right kidney. Decapsulation and nephrostomy of left kidney.	Recovery. Left kidney functioned soon after operation and right kidney began to function three days later.
III	F.	12	Appendicectomy.	9th day.	Pain in back. Hæmaturia.	280 cubic centimetres of 2·5% glucose in isotonic saline solution followed by 738 cubic centimetres of isotonic sodium sulphate solution intravenously.	Recovery. Passed 60 cubic centimetres of urine ten hours after receiving sodium sulphate solution, a further 60 cubic centimetres five hours later, and 2·8 litres twenty-four hours later.
IV	F.	11	Appendicectomy.	6th day.	Pain and scalding on micturition. Tenderness in both loins. Hæmaturia. X-ray revealed no abnormality. Convulsions.	300 cubic centimetres of normal saline solution intravenously. Nephrostomy of right kidney on sixth day. Nephrostomy of left kidney on tenth day. Both kidney pelves found tensely distended at operation.	Recovery. Right kidney functioned soon after operation. Left kidney functioned only after second operation.
V	F.	5	Appendicectomy.	7th day.	Frequency of micturition. Hæmaturia on fourth day after operation.	Diathermy to loins. Decapsulation and nephrostomy of both kidneys on eighth day. Both kidney pelves found tensely distended at operation.	Recovery. Both kidneys began to function soon after operation.
VI	M.	3½	No operation.	—	Incessant vomiting. Abdominal pain for three days, then anuria. Tenderness in both loins for forty-eight hours before operation of pyelostomy.	Nephrostomy of right kidney only. Pelvis of this kidney found to be tensely distended. Ureteral catheter passed down ureter met with obstruction at upper end.	Recovery. Both kidneys began to function soon after operation, which was performed on one side only.
VII	M.	2	No operation.	—	Screaming attack, abdominal pain and vomiting. Hæmaturia. Anuria for forty-eight hours and possibly longer before operation of pyelostomy.	Nephrostomy to left kidney. Pelvis found to be tensely distended. Ureteral catheter met with obstruction a few inches from uretero-pelvic junction.	Recovery. Left kidney functioned soon after first operation. Right kidney began to function only after second operation.
VIII	F.	15	Nephropexy.	16th day.	Vomiting and pain in right groin. Had had similar attacks before on left side. Both ureters catheterized. Convulsions seventeen days after operation. X-ray film revealed slight dilatation of the calyces on the left side.	Nephrostomy to right kidney forty-eight hours later; 900 cubic centimetres of 4% glucose solution given intravenously. Spinal anæsthesia and catheterization of both ureters.	Recovery. Forty ounces of urine drained through catheters in twelve hours.
IX	M.	10	Setting fractures of femora.	—	Hæmaturia.	Decapsulation of both kidneys with excision of segment of cortex for histological examination. Result showed that this operation was really a nephrostomy.	Recovery. Within twenty-four hours of operation five ounces of urine were voided and profuse urinary drainage from both lumbar incisions followed.
X	F.	60	No operation.	—	Sudden severe pain on right side, with vomiting. Had had similar attack on left side some time before. Right ureteric orifice red and œdematous. Left ureteric orifice normal, but no efflux seen.	Four pints of normal saline solution intravenously. Later ten cubic centimetres of hypertonic saline solution intravenously.	Recovery. Seventy-four ounces of urine voided on fifth day. Later passed small oval stone. Left kidney, as shown by excretion pyelogram, was not functioning when patient left hospital. Evidently there was still a stone blocking the left ureter.

Uebelhör[18] reports a number of cases of anuria, two following kidney trauma and several due to calculi which could be demonstrated only on one side. He states that "even today the possibility of reflex anuria is universally granted"; but the cases reported by him do not include any in which one could definitely exclude a bilateral lesion—and incidentally, he did not report any following appendicectomy.

The Treatment of So-Called Surgical Anuria.

After consideration of the cases reported, definite rules can be formulated as to the correct treatment. First, decapsulation alone is quite useless, for when it has been followed by recovery, some other factor has been responsible for the recovery. Ureteral catherization may effect a cure, but only when the catheters can be pushed past the obstruction into the kidney pelvis, and so lead to drainage of the pelvis. In most cases in which it has been attempted it has failed.

The only certain method of draining the pelvis is by pyelostomy or nephrostomy, and this procedure has been described above. Intravenous injections of glucose, saline or sodium sulphate solution may occasionally be successful, because they raise the pressure within the kidney pelvis, and so may lead to the displacement of a small calculus in the ureter. Although not usually successful in this way, the injection of glucose and saline solution is an important adjunct to treatment; this procedure stimulates diuresis after the obstruction has been relived, and so helps in eliminating toxins from the blood.

Summary.

1. A case of so-called reflex anuria is reported.

2. A number of similar cases which have been reported in medical literature, including three by the author, are critically reviewed.

3. Evidence is adduced to show that all these are cases of the well-known condition of calculous anuria.

4. It is suggested that reflex anuria has no existence in fact, and that the term should be abandoned.

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Reports of Cases.

PULMONARY TUBERCULOSIS TREATED BY ARTIFICIAL PNEUMOPERITONEUM.[1]

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THE establishment of artificial pneumoperitoneum is a simple and safe procedure which has been practised for a good many years with some success in the treatment of intestinal tuberculosis. It has been found that it may exert a beneficial effect upon pulmonary tuberculosis also, and during the past five years a number of articles have appeared in medical journals on the history, rationale, indications, technique and results of the procedure. Reference is made in the bibliography to three of the best of these articles. The present report of the case of a patient treated for pulmonary tuberculosis by means of artificial pneumoperitoneum may be of some interest, because the method may be, as here, the only practicable form of active treatment, and it has not so far been much, if at all, used in this country.

Clinical Record.

The patient was a woman, aged thirty-three years. At the age of twenty-six years she had had an operation performed for retroversion of the uterus, and shortly afterwards suffered from "pleurisy". In the following year she had hæmoptysis and entered the Royal Prince Alfred Hospital, where tuberculosis was diagnosed and an artificial pneumothorax was established on the right side. The pneumothorax was maintained for fourteen months, when refills became impossible owing to obliteration of the pleural cavity. At the age of thirty-two years she went into a decline; she had fever, troublesome cough with four to six ounces of sputum containing tubercle bacilli every day, and lassitude; and her weight fell from eight and a half to six and a half stone. In this state she was admitted to the Randwick Auxiliary Hospital. X-ray examination revealed that the upper lobe of the right lung was collapsed and contained a large cavity; there was also evidence of tuberculosis in the right lung just below the collapsed part, and very slight involvement of the upper zone of the left lung.

As rest in bed and good nursing did not quickly effect improvement in her condition, which in fact deteriorated further, an attempt was made to reestablish the artificial pneumothorax on the right side, but in vain, as was half expected. Major surgical measures were out of the question owing to her debility, and so the next move was to crush the right phrenic nerve; this was done by Dr. M. P. Susman, the honorary surgeon, and a satisfactory paralysis of the right side of the diaphragm was effected. During the next seven months the patient held her ground and gained three pounds in weight; but the X-ray appearances of the lungs were unchanged, and all the time she had slight pyrexia and produced four to six ounces of sputum a day. Then she relapsed, was much more pyrexial and became paler and thinner.

It was decided to establish an artificial pneumoperitoneum. The object was to raise the diaphragm, especially the paralysed leaf, and thus to effect some sort of collapse therapy for the tuberculous process about and below the large cavity in the right lung. The apparatus used was the same as for the establishment of artificial pneumothorax. The needle used was a Saugmann's needle with a terminal aperture and with an accurately fitting obturator instead of a stilet. Such a needle is very satisfactory for commencing as well as for refilling a pneumothorax. The puncture was made very close to the costal margin in the line of the lateral border of the left rectus muscle; this is the site of election, because elsewhere the abdominal wall yields before the needle and here no important structures are likely to be pierced. When the first puncture was made the fluid in the manometer did not oscillate with each respiration; but a small oscillation about the zero level occurred with each beat of the aorta. An amount of 200 cubic centimetres was introduced, and while it was flowing in, a small oscillation of the manometer occurred with each respiration. There was no doubt that the air was introduced into the peritoneal cavity, because the liver dulness was abolished anteriorly. The patient experienced no discomfort

[1] Presented at a meeting of the Section of Medicine of the New South Wales Branch of the British Medical Association on July 11, 1940.

at the time of the operation, but during the next week or so she had a good deal of abdominal and scapulo-humeral discomfort, especially on the left side. She insisted that this was only discomfort and not pain. The scapulo-humeral element was relieved when she lay flat in the bed for a time. Refills were carried out every few days at first, and after six weeks or so the interval between the refills was increased to a fortnight. The air in the peritoneal cavity was always found to be under a pressure greater than the atmospheric pressure, and there was very little fluctuation of the pressure with respiration; further air would not enter the peritoneal cavity from the apparatus until it was driven in under a much higher pressure than that registered on the manometer, and at the end of the operation the intraperitoneal pressure was very little if at all higher than at the beginning. For this reason it was often hard to be sure from observation that the point of the needle was in the peritoneal cavity; but if the intraperitoneal pressure was increased by voluntary muscular action this increase could be registered on the manometer.

The patient's health began to improve as soon as the pneumoperitoneum was established. She felt better and brighter at once and her appetite greatly improved. The fever gradually became less, and the amount of the sputum was reduced to one and a half to two and a half ounces a day. The change for the better in her appearance was conspicuous. An X-ray film showed that the diaphragm was raised almost an inch on both sides and that the heart was displaced upwards and tilted to the right (Figures I and II).

Unfortunately this pleasant story has a sad sequel. Although the patient was so much better, there were still the large cavity in the right lung and the bacilliferous sputum, and it seemed that her prospects of getting up and about again were remote, even if no complications arose to vitiate the efficacy of the pneumoperitoneum. It was necessary to contemplate major surgical procedures to close the cavity as soon as she should be well enough to stand it. At the end of four months she seemed fit to undergo the first stage of a thoracoplasty. Dr. Susman was asked to see her and agreed to operate; but the patient absolutely and very firmly refused this treatment—she would submit to anything but thoracoplasty. Accordingly Dr. Susman was asked if he would consider establishing an extrapleural pneumothorax; but he replied that he did not feel that it would be right to perform an operation that was not the operation of choice, and there the matter rested. However, after a few weeks, as the patient still refused thoracoplasty and as hers was regarded as rather a special case, the operation was undertaken; unfortunately the cavity was opened (the chief hazard of establishing extrapleural pneumothorax over a large cavity) and the patient died a few weeks later.

Post-Mortem Findings.

An autopsy was made. The peritoneum was found to be normal in all respects to the naked eye and on microscopic examination. The puncture marks could not be recognized. There were a few air bubbles under the capsule of the right lobe of the liver and a few adhesions in the true pelvis. The musculature of the right (formerly paralysed) side of the diaphragm was indistinguishable from that of the left side.

Acknowledgement.

The Director-General of the Department of Public Health of New South Wales (Dr. E. S. Morris) kindly permits me to report this case.

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LABOUR COMPLICATING FEBRILE GASTRO-ENTERITIS.

By MARY C. DE GARIS.

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Clinical Record.

A SINGLE woman, aged twenty-four years, first attended the antenatal clinic when seven months pregnant; she paid in all four visits to the clinic. The patient was a *primipara* who had had one miscarriage, which was said to have been septic. She was edentulous, her diet was good and the menstrual periods had been painless. She was admitted to hospital on April 6, 1939; vomiting and diarrhœa had been present for the past week. Her temperature was 99·4° F. and she was not in labour, although the expected date of her confinement was fourteen days past.

The patient was given castor oil and bismuth; her temperature rose to 100·2° F. and labour began. The fœtus was born in the right occipito-anterior position on April 7, 1939. The first stage of labour lasted fifteen and a quarter hours, the second stage four hours and fifty minutes, and the third stage two hours and fifteen minutes. A catheter specimen of urine taken during the second stage of labour contained a trace of albumin and a few hyaline casts, but no leucocytes. The patient was delivered of a living female infant, weighing eight pounds five and a half ounces. The baby was weaned after a few days. Severe post-partum hæmorrhage necessitated the manual removal of a "battledore" placenta adherent over two-thirds of its surface. Much treatment was required for the acute anæmia; the patient was given hæmostatic serum, submammary and rectal injections of saline solution, and later a transfusion of blood. Two other blood transfusions were given during the puerperium, on April 11 and 15, and also another dose of hæmostatic serum. The vomiting and diarrhœa persisted for a few days; on April 9 the lochia became offensive. On April 11 the hæmoglobin value was 25%, on April 12 it was 30%, and on April 18 it was 40%.

The patient's temperature was coming down, but it suddenly rose to 104·2° F. on the fifth day of the puerperium, to 104·6° F. on the twenty-seventh day, and to 104·8° F. on the thirty-fifth day. On April 11 no pyuria was present, but the urine was albuminous and highly acid, while on April 24 and May 10 pyuria was present, associated with the sudden rises of temperature. The patient was treated as suffering from recurrent pyelitis; she was also given "M & B 693", and her temperature came down. A little pus was present in one of the blood transfusion wounds. She was discharged from hospital on the fifty-fifth day, her temperature having been subnormal for sixteen days.

Discussion.

This case was regarded as an instance of autogenous infection; the febrile gastro-enteritis was, of course, an accident, while the pyelitis may have been secondary to it, though more probably it was the common pyelitis of the puerperium exaggerated by the lowered resistance due to the acute anæmia.

It is of great clinical importance to recognize the pyelitic character of much of the fever of the puerperium, for pyelitis responds readily to treatment; but if it is neglected it easily passes into true septicæmia. Diagnosis of the character of a puerperal fever, whether accidental (carried) or autogenous, is as necessary as in other fevers.

Reviews.

DISEASES OF THE DIGESTIVE SYSTEM.

FROM time immemorial diagram has been the means *par excellence* for the communication of an idea, and modern educationists are insisting more and more upon the value of visual education as a method of instruction.

Dr. Eugene Rosenthal,[1] who is a teacher of distinction in the first medical clinic of the Royal Peter Pazmany University at Budapest, presents a technique which, although dogmatic and eclectic, relies largely for its efficacy upon a large series of geometrical tables and of coloured diagrams (some rather reminiscent of "Neon" signs) in lieu of the customary catalogue of facts and theories. For well over a decade a somewhat similar method has been in vogue in such widely separated centres as the Deutsches Hygiene Museum at Dresden, the Wellcome Foundation Museum in London, and the Museum of Hygiene and Medicine of the Mayo Clinic at Rochester, United States of America.

This realistic and vivid presentation of pathological processes has recently been installed into the Kitchener School of Medicine at Khartoum and might profitably be initiated into the medical schools of our own Australian universities.

[1] "Diseases of the Digestive System: A Text-Book for Students and Practitioners", by E. Rosenthal, M.D., with a preface by R. J. V. Pulvertaft, M.D., F.R.C.P.; 1940. London: Henry Kimpton. Crown 4to, pp. 406, with 234 illustrations, including 104 in colour, and 16 tables. Price: 42s. net.

The above-mentioned volume, however, is much more than a mere scientific "picture book", for many vexed questions in gastro-enterology are logically and lucidly discussed.

The second chapter is given up to a comprehensive review of the various clinical methods utilized in the making of an accurate gastro-intestinal diagnosis. If diarrhœa is a prominent symptom, the test diet of Schmidt and Strasburger should be administered over three days and the stool passed on the fourth morning is then submitted to a most meticulous and thorough examination in an endeavour to assess the functional activity of the intestine and its various associated glands—for example, in the differential diagnosis of "fermentative dyspepsia" and "putrefactive dyspepsia".

The frequency of chronic gastritis is then referred to, and, although the part played by exogenous factors—mechanical, chemical, thermal and bacterial—in producing inflammatory changes is stressed, no reference to the more recent work of Knud Faber on the part played by hæmatogenous infection is encountered until the section on achylia is reached.

Although the author states that achylia should be regarded as a special form of gastritis, the reader is somewhat surprised to find that the descriptions of the two maladies are divorced by the intervention of some fifty odd pages.

In the section dealing with "Gastric and Duodenal Ulcer" the author points out that any dietetic régime should be based upon retrogression of the ulcer; hence the conservative or medical treatment is divided into four stages. The first stage terminates when symptoms are in abeyance; there is an absence of occult blood from the fæces, and fluoroscopic examination reveals a diminution in the size of the ulcer. The third period commences when the crater has been reduced to one-half and terminates with the complete disappearance of the ulcer shadow. The fourth or convalescent period is comparable with the real test of cure. If such treatment fails to secure progressive reduction in the size of the ulcer, the author, notwithstanding the fact that continuous gastric alimentation has been conclusively proved to be much superior both objectively and subjectively to Einhorn's method of continuous duodenal feeding, unhesitatingly advocates transduodenal feeding.

Few gastro-enterologists would subscribe to the theory that olive oil "forms a protective layer on the gastric mucosa and the food then evokes a smaller secretion of acid".

The disadvantages of alkaline therapy are enumerated, and such therapy is summarized as "a necessary evil that cannot be dispensed with". The author stresses the statement that if atropine is prescribed it should be administered hypodermically and in much more liberal doses than are customary in British communities.

The reader will find much to interest him in the presentation of appendicitis and intestinal obstruction, and these sections must be regarded as one of the most satisfactory reviews in the English language.

The second half of the book is devoted to diseases of the liver, biliary passages and pancreas, and opens with an excellent summary of the commonly used bedside clinical observations and laboratory investigations which may be used in the investigation of this group of diseases.

The section on cholelithiasis is terse, highly critical and intensely stimulating. The infective (Naunyn's), mechanical (Aschoff's), hypercholesterolæmic (Chauffard's), and hepatogenous-bilihumin nuclear (Boysen and Rovsing's) theories are dealt with *seriatim* and the many imperfections of each are forcibly portrayed. The author succinctly summarizes the ætiology thus: "Each of these explanations seemed in their day to be satisfactory, but the ultimate cause of stone formation is still far from certain."

A gall-stone carrier becomes a gall-stone sufferer through the development of inflammatory changes in the biliary tract. The author then contrasts the medical treatment of inflammatory affections of the urinary tract with those of the biliary passages and goes on to state:

> No special diet exists for the treatment of cholelithiasis—the stereotyped principle of the restriction of fats cannot be supported for various reasons. Medicines occupy a certain place in the treatment of gall-stones. I have never succeeded in demonstrating formaldehyde in the bile recovered from the duodenum after the intravenous injection of urotropin.
>
> Further after intravenous administration Salicyl appeared abundantly in the urine, but no trace of it was demonstrated in the bile.
>
> These instances have been quoted only to show that many of the widely known curative methods are of rather doubtful effect.

The volume concludes with a clear, concise and thoroughly up-to-date account of the more modern methods which may be employed in the making of an accurate diagnosis in diseases of the pancreas, due emphasis being laid on the many pitfalls that may be encountered in their practical application.

This new and attractive text-book should meet a definite need. Perhaps not every reader has a strong visual memory; but those who have will find it hard to forget the unique diagrams so lavishly presented.

A MANUAL OF EMBRYOLOGY.

WHEN the first edition of Frazer's "Embryology" appeared about ten years ago, it was regarded as a distinguished achievement in British embryology. In addition to the fact that as a manual of embryology based on the original work of the author it was the first of its kind to appear in Britain, the text obviously represented a masterly handling of the subject.

The author's description of the early stages of human development and the original figures which accompanied it were amongst the best yet published in any language, and copies of some of the figures soon found their way into other well-known text-books.

In a book of such uniform excellence, however, it is difficult to pick out any particular section for special comment. If any such attempt were made, the sections on the development of the brain, pharynx and gut deserve special mention. These are particularly well written and elucidate difficult chapters in embryology.

In the new edition the author has taken the opportunity of rearranging some parts of the text and of adding some new figures and material; but on the whole the plan of the book is the same. Since its first appearance the book has been a constant source of help and inspiration to teachers and students alike, and the present revision will add to its usefulness.

SURGERY OF THE HAND.

"SURGERY OF THE HAND", by Marc Iselin, of Paris, is the first volume of a two-volume work.[2] The author calls it the practitioner's book, as distinct from the surgeon's book, which was to come later and deal with the more advanced problems of operative surgery. The book is written with a Gallic prolixity and redundancy, and in parts it partakes more of the nature of a research thesis than of a text-book.

It is interesting to note that the author condemns passive hyperæmia and hand-baths—the first as useless, the second as harmful, partly on account of the gravity of secondary staphylococcal infections. He finds antitetanic serum of doubtful value. Perhaps the two most stimulating suggestions in which he may improve upon current practice are his advocacy of immobilization of the infected or injured hand until the wounds are healed, and his method of opening only the superior *cul-de-sac* of infected tendon sheaths. Most surgeons would condemn many of his radical methods—for example, the excision of all necrotic tissue in the felon, resection of the finger and wrist joints for injuries and infections of these joints, and the driving of a nail through the metacarpal head for fractures of the shaft.

The author inaccurately represents Kanavel's incisions for teno-synovitis, and criticizes one because it menaces the digital sensory nerve. In his own 1931 incision for the radial sheath the much more important motor nerve to the thenar eminence is quite forgotten.

In discussing treatment he sometimes overlooks the importance of elevation; in acute teno-synovitis he does not mention the possible value of sulphanilamides; and in perungual whitlow he has not recognized that satisfactory drainage can sometimes be achieved by lifting the eponychium with the blunt end of a needle. For cut flexor tendons he decries immediate operation (unlike Bunnell) and recommends that the wrist and fingers shall be kept in full flexion for three weeks. Böhler holds that this position, maintained for over a week, will result in prolonged or permanent crippling in patients over forty. The author's discussion on the punctured wound is inadequate, hard to find and not mentioned in the index.

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[1] "A Manual of Embryology. The Development of the Human Body", by J. E. Frazer, D.Sc., F.R.C.S.; Second Edition; 1940. London: Baillière, Tindall and Cox. Royal 8vo, pp. 533, with 282 illustrations. Price: 30s. net.

[2] "Surgery of the Hand: Wounds, Infections and Closed Trauma. A Book for the Practitioner and the Surgeon", by M. Iselin, M.D., translated by T. M. J. d'Offay, M.B., Ch.B., F.R.C.S., and T. B. Mouat, Ch.M., F.R.C.S.; 1940. London: J. and A. Churchill Limited. Demy 8vo, pp. 366, with illustrations. Price: 21s. net.

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THE ULTIMATE PROGNOSIS AND REHABILITATION IN CORONARY OCCLUSION.

UNTIL a few years ago coronary occlusion was generally regarded as a hopeless condition, and there are many today, particularly among the laity, who look at it in the same way. Those who have had experience of the malady have always recognized that recoveries do occur and that certain persons may, if sufficient care is taken of them, be guided along the path of convalescence until they are able to take their places again as apparently healthy citizens. Anatomical and pathological studies have led to a fuller understanding of the condition, and clinical experience has shown a much wider circle of practitioners than was previously acquainted with it that the outlook of a stricken person is by no means entirely black, provided he can survive the initial attack. The aspect of a person in the throes of an attack and the symptoms are so well known that the patient and his relatives are certain to be alarmed, if not panic-stricken; the medical attendant must therefore be conversant with the significance of the symptoms and with their possible bearing on the patient's future.

Reference to this subject has been prompted by the appearance of a study by A. M. Master and Simon Dack of a series of patients who had sustained acute coronary occlusion and had survived the attack.[1] Before this report is discussed, however, it will be well to recall some of the main features that have a bearing on the ultimate prognosis. To begin with, a large number of patients die during the early and acute stage of the illness. The mortality rate has been variously estimated, but most writers put it down at something like 50%. The survivors may be divided into two groups. The first includes those whose initial attack is followed by an increasing tendency to dyspnœa due to progressive pulmonary œdema and a drift towards congestive heart failure; these people may live for a month or two. The second group includes those who make a satisfactory recovery. It is important that the clinician should have at the start a clear mental picture of the difference between coronary occlusion—total obliteration of the lumen of the vessel—and so-called coronary insufficiency, due to partial occlusion or spasm. In some ways it is a pity that the word insufficiency has passed into such general use in this connexion; it is clearly not quite suitable, for total occlusion may also be described as insufficiency. It must also be remembered that the occurrence of a cardiac infarct may be merely a phase of a generalized arterial disease; in other words, patients with a cardiac infarct do not always die of cardiac disease, but may die of cerebral hæmorrhage or renal failure. When a coronary vessel becomes occluded, the area of heart muscle that is deprived of its circulation undergoes necrosis and the fate of the patient will depend on the size of the area involved, as well as on other factors. It has been pointed out that the occurrence of a cardiac infarct may be merely a phase of a general disease, but blockage of a coronary vessel is most often a matter of almost local arterial disease—it is due to atheroma rather than to general arteriosclerosis; this is shown by the large number of patients with coronary occlusion who have no hypertensive disease. After the occurrence of occlusion, if the patient does not die of ventricular fibrillation or failure of the circulation from some other cause, scar tissue is gradually formed in the necrotic area, and if the area is not too extensive and the condition of the remaining heart muscle and of the vessels is satisfactory, the scar tissue may become strongly organized and the heart wall be capable of carrying on its function. The coronary arteries are not end arteries, as was once thought; collateral circulation, though not particularly extensive, does occur, and strangely is more likely to develop in the heart muscle of a subject who is past middle life than in the muscle of a younger person. This anastomosis with the formation of strong scar tissue is essential to recovery. For the sake of completeness, mention should perhaps be made at this stage of the possibility of the development of cardiac aneurysm when the infarction has involved the wall of the left ventricle. This is a well-recognized condition and is the result of stretching of the scar tissue resulting from infarction; a discussion and an account of the clinical manifestations, together with details of cases in which a diagnosis was made during life, will be found in an article recently published by W. Dressler and R. Pfeiffer.[1] When an attempt is made to determine the future prospects of a patient who has recovered from the initial attack of a coronary occlusion, all available data must be used, for no single criterion is sufficiently reliable. East and Bain, in "Recent Advances in Cardiology", point out that those patients do best who recover steadily from the onset. This is to be expected. J. Cowan has stated that enlargement of the heart is of bad significance, whether it is due to hypertrophy or dilatation; the former indicates a preexisting strain on the heart and the latter a failure of the surviving muscle to compensate the loss of some muscle cells. Curiously, the electrocardiograph is not of much assistance. If after recovery from a coronary occlusion the electrocardiogram should return to normal, this would be regarded as a most favourable sign; but

[1] *The Journal of the American Medical Association*, September 7, 1940.

[1] *Annals of Internal Medicine*, July, 1940.

satisfactory progress may be made by the patient without a return to normal in the electrocardiographic findings.

Turning now to the report of Master and Dack, to which reference has already been made, we find that they dealt with 415 patients who had survived an acute coronary occlusion. Of the 415 patients, 342 were males and 73 females; 75% had suffered one attack, 22% a second attack, and the remainder a third or fourth attack when they first came under the observation of the authors. In the available space it will not be possible to give a full account of the observations made on these persons; nor is it necessary to do so, for we are at the moment concerned with general principles rather than with details. Those who are interested in cardiology are urged to study this paper at leisure. Master and Dack found that approximately half the number of patients were able to resume their former occupations within one year and usually in less than six months. Many of those who returned to work complained of angina, dyspnœa or weakness; but these symptoms were usually mild and often no more severe than they had been prior to the attack. Many of the patients were observed at regular intervals, and Master and Dack gained the impression that as a rule the symptoms were not directly connected with the fact that the patients were employed, but would have been present in any case. Only occasionally was it found that while a patient was employed symptoms would develop which would disappear when he gave up his job. Master and Dack are well-known workers in this field, and in conjunction with H. L. Jaffe have recently produced evidence to show that effort plays no part in the causation of coronary occlusion.[1] Their observations in the present study are in keeping with the views put forward by themselves and Jaffe. They place patients who recover from coronary occlusion in two main groups. The first includes those who recover quite well and who can resume their usual mode of life, though they may have some complaints. The great majority of this group are patients below the age of fifty years, who have sustained only one attack of coronary occlusion. In the other group are those who do not completely recover from the occlusion, but are left with a persistent anginal syndrome or evidence of heart failure; these are not able to return to work. In regard to angina, one or two interesting observations may be made. In the first place anginal pain does not always occur in coronary occlusion. H. M. Pollard and T. H. Harwill, in a study of 375 cases of myocardial infarction, found 17 instances of undoubted coronary occlusion in which no pain, substernal pressure or other anginal symptoms had occurred at any time; among the symptoms that occurred at the time of the accident the commonest were dyspnœa, nausea and vomiting, dizziness and fainting or collapse.[2] The second observation is the well-known fact that some patients suffering from acute anginal pains lose their angina completely after occlusion has occurred. Lewis explains this by suggesting that the original pain arises from an area supplied by a narrowed artery, that this vessel becomes thrombosed and that the pain-giving muscle dies.

When Master and Dack state in their general conclusions that an attack of coronary thrombosis is in itself not sufficient reason for permanent disability, they make a valuable pronouncement that must be interpreted in the light of the considerations that have been stated above. There is no doubt that the additions that have been made to the knowledge of this subject during recent years have given to the patient recovering from an acute attack of coronary thrombosis much more hope than he used to have. There is no need for him to be classed among those who are without hope in this world and therefore "of all men most miserable".

[1] *Industrial Medicine*, July, 1940.
[2] *The American Journal of the Medical Sciences*, May, 1940.

Current Comment.

STUDIES ON PRESERVED BLOOD FOR TRANSFUSION.

THE occurrence of a war, creating the need for well-organized facilities for emergency blood transfusion, has stimulated tremendous interest in the use of blood that has been kept stored for just such an emergency. It was indeed on active service towards the end of the last war that stored blood was first used for this purpose. A number of studies on the storage and preservation of blood have recently been described. One of the most comprehensive is that by E. L. De Gowin and R. C. Hardin.[1] It is found that when blood is stored a certain degree of hæmolysis occurs, no matter what the preservative used. The rate of hæmolysis is much greater when blood is stored at 20° C. than at 5° C. The addition of large quantities of isotonic dextrose solution to blood slows considerably the rate of hæmolysis as compared with that in blood stored with little or no dextrose. The solution found most satisfactory by these workers was ten volumes of blood, thirteen volumes of 5·4% aqueous solution of anhydrous dextrose and two volumes of 3·2% dihydric sodium citrate in water. This meant that to give 500 cubic centimetres of blood a total volume of 1,250 cubic centimetres was required. The dextrose, however, is usually no disadvantage, and the concentration of citrate is not high enough to cause any ill effects. Blood stored in this dextrose-citrate mixture will hæmolyse only one twenty-fifth to one-fiftieth as much in thirty days as blood stored in sodium citrate alone or in sodium citrate and sodium chloride. This mixture De Gowin and Hardin have found safe and practical in transfusions to humans. It is of more than ordinary interest, however, to find F. Corelli[2] using as a preservative sodium thiosulphate, another innocuous substance. He adds 7·5 cubic centimetres of a solution of this substance to each 100 cubic centimetres of blood, which then keeps good for three weeks. He has given one thousand transfusions with this mixture, 92% with no reaction at all and the remaining 8% with nothing but a mild rise of temperature. These results must be regarded as highly satisfactory.

Another factor increasing hæmolysis in stored blood is contact between the blood and air. The blood should therefore be stored in sealed flasks from which the air has been completely displaced by the blood mixture. To use an air-tight rubber stopper but to leave a small pocket of air in the flask is no more effective than merely to use a cotton wool plug. Agitation causes some hæmolysis of stored blood; but this is less in the dextrose-citrate mixture than in a simple citrate or citrate-saline mixture. The preservative is cooled to 5° C. before the blood is added, so as to minimize the initial hæmolysis, which is quite considerable if warm blood is added to the uncooled preservative.

A further factor of importance in stored blood is the diffusion of potassium that takes place from the cells to the plasma. The rate of this diffusion could not be decreased by varying the amounts of citrate, dextrose, sodium or chloride in the mixture. However, this high

[1] *The Journal of the American Medical Association*, March 9, 1940.
[2] *Acta Medica Scandinavica*, February 13, 1940.

potassium content of the plasma causes no ill effects if the rate of transfusion does not exceed 43·3 cubic centimetres per minute. The concentration of potassium in the plasma of stored blood is not sufficient to raise significantly the level of potassium in the recipient's plasma. J. Scudder and K. Bishop[1] have described an ingenious flask consisting of an upper and lower chamber separated by a narrow neck. The volume of each chamber is of such a size that when the red cells settle into the lower chamber the interface between cells and plasma lies in this narrow neck. The rate of diffusion between cells and plasma of the cell potassium is thus materially decreased. A similar decrease of diffusion of hæmoglobin also occurs.

The blood is stored at a temperature very much below body heat, and in an emergency valuable time may be lost if the blood is warmed before being given. De Gowin and Hardin have therefore investigated the effect of transfusing cold blood and other fluids. A detailed study of ten patients who were given transfusions of blood at temperatures ranging from 15° to 25° C. at velocities from 6·0 to 42·8 cubic centimetres per minute revealed no significant lowering of body temperature, no consistent changes in blood pressure, and no untoward clinical symptoms. After all, it causes no harm to hold a limb in cold water and so to send a stream of chilled blood back to the body. Cold preserved blood has been given to 568 patients by these workers with no reactions that could be attributed to that cause. The transfusion of cold stored blood is a great convenience in emergency work, and indeed reduces the risk of hæmolysis in the stored blood. Whether the hæmolysis is just as great when the blood is warmed in the body as it would have been had the warming taken place in the flask is not considered. At any rate, the significant thing is that no harm came to the patients. Nor were the reactions from any other cause more frequent with stored blood. Provided proper care was taken in the storage and handling of the blood, the reactions were no more frequent or more severe than when fresh blood was used.[2] Variation in the age of the blood caused no corresponding variation in the incidence of reactions. No types of reactions occurred from preserved blood which are not recognized as complications of transfusions of fresh blood.

It has also been determined that the life of neutrophile leucocytes is limited in stored blood. The level of prothrombin has been found to fall steadily, to reach about 40% of the original level at the end of three weeks.[3] Thereafter it remains about the same. The prothrombin level of stored blood was discussed in this journal on September 14, 1940. Stored blood is therefore of less value than fresh blood to control the bleeding of jaundice or hæmorrhagic disease of the new-born. The significance of prothrombin in this latter disease was discussed in these pages on September 7, 1940.

In view of the above findings it is interesting to find that the Johns Hopkins Hospital blood bank, as described by M. Ravitch,[4] stores simple citrated blood. No mention is made of the amount of hæmolysis occurring, but the time limit set for storage is ten days, and most of the blood is used in three to four days. Ravitch describes in detail the organization and technique of a blood bank which is functioning satisfactorily, and is not concerned with an analysis of results or reactions. His description is comprehensive, and many useful suggestions will be found by those interested in the establishment of a blood bank. We do think, however, that the equipment described, with its vent tubes and ball-bearing filters, is unnecessarily complex, and that the admirable equipment described by Hipsley in this journal of November 16, 1940, is simpler and cheaper, and could be equally well used with a bank. Indeed it can be used with the utmost ease for single or continuous infusions of any fluid desired. In this report from the Johns Hopkins Hospital, the discovery that separate banks of Negro and white blood are kept causes an instinctive sense of surprise. "Although there is, of course, no valid objection on biologic or physiologic grounds to the transfusion of patients of one race with blood from donors from another, it has been deemed best to avoid the issue." It requires little thought to realize the significance of the issue; but it provides a commentary on the depth of race consciousness in America. Lest we be charged with unjustified national pride, we admit that a similar colour prejudice, foolish and uninformed as it may be, would probably be found among most Australians in similar circumstances. Another feature of the report that calls for comment is the statement that in order to ensure a quick turnover of the blood in the bank, it has been made compulsory for all ward transfusions to be performed with bank blood, even though a compatible donor is readily and immediately available. With this rule we cannot agree, even though exceptions are made in certain diseases, the chief of which are leuchæmia, granulocytopenia and purpura. Transfusions of stored blood have proved safe, and for most diseases therapeutically valuable, but it is known with certainty that neutrophile leucocytes and prothrombin degenerate and diffusion of potassium occurs in stored blood. Some hæmolysis occurs and fibrin too sometimes separates out. It is possible that similar deterioration takes place in other elements, for blood is a very complex substance, and the significance of each of its constituents is not yet fully comprehended. We do not for a moment deny the value and importance of a blood bank for emergency work, but we do regret that its maintenance should mean as a rule the discouragement of the use of fresh blood when this is available.

[1] *The Journal of the American Medical Association*, July 27, 1940.
[2] *Ibidem*, September 14, 1940.
[3] *Ibidem*, April 6, 1940.
[4] *Ibidem*, July 20, 1940.

CÆSAREAN SECTION PERFORMED POST MORTEM WITH A LIVING CHILD.

THE operation of Cæsarean section has been performed on the dead mother since the earliest times, possibly even, De Lee tells us, by the early Egyptians. The operation is referred to in the myths and folk lore of European races. Dionysus was cut from the dead Semele. The *Lex Regia* of Numa Pompilius, 715 B.C., required that the child should be removed before the burial of the mother; and in 1608 the Senate of Venice laid it down that any practitioner who failed to perform Cæsarean section was liable to heavy penalties. The operation is not often performed nowadays on a dead woman, and when it is performed the child more often than not is lost. If the child is to be saved it must be removed from the uterus within a few minutes of death, and the chance of survival will obviously depend on the condition of the mother just before death. If the mother's death is sudden or if she dies of apoplexy or of an acute condition, the child is likely to live much longer than if the mother's death agony is prolonged. Since the mother uses up the child's oxygen the child is likely to die first. De Lee states that a fœtus will live for a period of from five to twenty minutes after the mother's death, and he adds that reported cases of longer survival are not authenticated. Joyce Morgan reports the performance of Cæsarean section on a woman, aged thirty years, who was admitted to hospital suffering from eclampsia and cerebral hæmorrhage.[1] She died four hours after her admission to hospital. She was seen a few minutes (two to seven) after death and the fœtal heart was heard to be beating. Section was at once performed and a live child was delivered; the child when a year old was healthy. Morgan states that one of her reasons for reporting the case is that opportunities for the performance of the operation *post mortem* may arise in the course of air raids, and the only instrument necessary is a scalpel or even a penknife. Apart from this aspect the case is of interest on account of survival when the mother was eclamptic.

[1] *The Lancet*, August 3, 1940.

Abstracts from Medical Literature.

MEDICINE.

Nicotinic Acid Deficiency Encephalopathy.

NORMAN JOLLIFFE *et alii* (*The Journal of the American Medical Association*, January 27, 1940) report a series of 150 cases of an encephalopathic syndrome which they believe is caused by nicotinic acid deficiency. The syndrome may occur as the only clinical manifestation of a deficiency disease, or it may occur in association with pellagra, with polyneuritis due to vitamin B_1 deficiency, with the oculomotor disturbances of a central neuritis or with scurvy. The clinical picture is more or less well defined and is characterized by a clouding of consciousness, by cogwheel rigidity of the extremities and by uncontrollable grasping and sucking reflexes. The first group of forty-seven patients were treated by hydration, infusions of 5% dextrose in normal saline solution being administered; 90% of these died. In view of the associated polyneuritis it was suggested that this syndrome might also be due to deficiency of one or more factors of the vitamin *B* complex. Thiamin chloride was therefore added to the treatment by hydration, 50 to 200 milligrammes being administered daily by the parenteral route. Fifteen consecutive patients thus treated all died without manifesting any improvement. The next group were treated with vitamin-rich diets supplemented by oral and parenteral administration of large amounts of preparations rich in vitamin *B*: brewer's yeast, aqueous whole liver extract and various fractions of liver. Of the 66 patients in this group, 33 had other signs, now recognized as due to nicotinic acid deficiency, namely, pellagra, stomatitis, the common psychic symptoms and dermatitis; only 51% of this group died. This is thought to suggest that the responsible factor must be sought in the vitamin *B* complex, with the exclusion of thiamin chloride. Twenty-two consecutive patients with the encephalopathic syndrome were treated with nicotinic acid; the mortality rate was 14%. This treatment was carried out as follows: hydration was produced by the oral administration of 180 cubic centimetres an hour of 5% dextrose in normal saline solution; if the patient was able to eat, a basal diet extremely low in the entire vitamin *B* complex was given. The first 16 patients were thus treated for three days before the administration of nicotinic acid, 500 milligrammes a day, divided into five equal doses and added to the dextrose saline solution: later this was increased to 1,000 milligrammes a day in ten divided doses. Finally it was decided to add 100 milligrammes daily of sodium nicotinate, diluted in 5% dextrose in normal saline solution and administered intravenously, and 100 milligrammes of sodium nicotinate given intramuscularly. When recovery from the syndrome occurred, usually in the third to the fifth day, a high caloric diet, rich in all vitamins, was given, supplemented by 18 grammes of "Vegex" and nicotinic acid given by mouth in small doses for a further two to ten days. Other deficiency features were then treated by the appropriate factors. It is pointed out that administration of nicotinic acid will not always result in cure, for the deficiency may have advanced to an irreversible stage. The authors discuss the possibility that this encephalopathic syndrome may represent a complete nicotinic acid deficiency, whereas the pellagra syndrome represents only a partial deficiency.

A Suggested Cure for Typhoid Carriers.

W. SAPHIR AND KATHARINE M. HOWELL (*The Journal of the American Medical Association*, May 18, 1940) report the case of a nurse, aged twenty-three years, who suffered from chronic diarrhœa, and from whose fæces *Bacterium paratyphosum A* could always be recovered over a considerable period of time. Bacteria were also found in the duodenal contents. Attempts were made, without success, to eradicate the bacteria by the administration of a vaccine, of sulphanilamide and of kaolin. One day a dose of four grammes of soluble iodophthalein was administered orally in the form used for cholecystography. On the following day bacteriological examination of the stools failed to reveal the organisms. Administration of the dye was repeated twice in the next six days, and though the stools were repeatedly examined during the next six months the bacteria were never once found.

Combined Doses of Zinc Protamine Insulin and Soluble Insulin.

G. M. WAUCHOPE (*The Lancet*, May 25, 1940) states that when zinc protamine insulin and soluble insulin are drawn into the same syringe, the action of the soluble insulin is modified, some of it being delayed in action and some of it apparently lost.

The Chemical Diagnosis of Acute Alcoholism.

WALTER W. JETTER (*The New England Journal of Medicine*, December 28, 1939) discusses the clinical and chemical diagnosis in 1,000 cases of acute alcoholism. The clinical criteria were (*a*) abnormality of gait, (*b*) any two of the following: abnormality of speech, the use of only familiar phrases, dilatation of the pupils, flushing of the skin, and alcoholic odour in the breath. The chemical methods of determination of alcohol in body fluids are discussed and the results are correlated with the clinical findings. The diagnosis was made clinically in 47% of cases with a blood level of 0·15 gramme per 100 cubic centimetres, in 83% with a blood level of 0·20 gramme *per centum*, and in 90% with a blood finding of 0·25 gramme. The actual blood alcohol concentration at which a person should be considered intoxicated would seem to depend on the definition used in the diagnosis of the condition. A great deal of experimental work has been done to prove that even small concentrations of alcohol in the blood (0·04 to 0·10 gramme per 100 cubic centimetres) slow the reaction time, impair judgement and increase the number of errors during the performance of mechanical tests. A blood alcohol concentration of 0·15 gramme per 100 cubic centimetres represents an appreciable consumption of an alcoholic beverage, as the average person weighing 70 kilograms must ingest six or seven ounces of 100% proof whisky within an hour in order to attain this level. The chemical test would do much to clear up many of the difficulties associated with the legal aspects of intoxication, for it shows whether the individual has been drinking and the approximate amount that must have been consumed. It is not suggested that the clinical features should be excluded; rather they should be considered as another means of arriving at a proper interpretation of certain physical and mental abnormalities which may or may not have been produced by alcohol.

The Effect of Sulphapyridine on the Urinary Chlorides in Pneumonia.

W. ANTOPOL AND J. CHURG (*The Journal of Laboratory and Clinical Medicine*, June, 1940) have investigated the excretion of chlorides in the urine by persons under treatment for pneumonia with sulphapyridine. In these patients the urinary chloride excretion was very small at the height of the illness, but a great increase occurred usually four or five days after defervescence. The authors point out that although sulphapyridine does not produce all the changes associated with spontaneous crisis, the increase in urinary chloride excretion occasioned by it appears to be analogous to that which occurs one to three days after spontaneous crisis.

Gastric Hyperacidity.

A. L. BLOOMFIELD (*The American Journal of Digestive Diseases and Nutrition*, December, 1939) discusses the problem of gastric hyperacidity and reports the results of his investigations of the gastric secretion. The term hyperacidity has been applied to highly acid gastric juice, to a very free flow of gastric juice, usually of high acidity, and to a group of clinical symptoms including fullness, epigastric distress and regurgitation of acid material. The subject has been confused ever since it was known that the stomach produced acid. Polland and Bloomfield in 1931, using a standard histamine test, measured the gastric secretion of persons having no evidence of stomach disorder and found a total acidity varying between 30 and 150; the majority had a total acidity of 110 to 120, but there were numerous exceptions at both ends of the curve. The highest quantity of gastric juice at the end of ten minutes after histamine stimulation varied between eight and 70 cubic centimetres. Polland also investigated 166 patients with peptic ulcer. In these were found the most acid and profuse gastric secretions, but in no case were the limits for normal transgressed. So that, after the administration of histamine, though a high or low acidity might be found, it was regarded as inaccurate to speak of hyperacidity. However, when the gastric juice was aspirated from a fasting patient at rest without histamine, the total acidity in normal persons was under 100; but in patients with duodenal ulcer a total acidity over 100 was frequently noted. In other words, an acidity in excess of normal occurred frequently in duodenal ulcer. The volume of gastric juice was also increased in these patients when this so-called basal secretion was investigated; the normal ten-minute volume was rarely more than 15 cubic centimetres, whereas patients with ulcer secreted 20 to 30 cubic centimetres in the same period. It was noted, therefore, that hyperacidity and hypersecretion usually coincided. However, patients with this excess did not necessarily have symptoms of indigestion. It has been demonstrated further that

sensations of acid or burning, suggesting acidity, can be produced by the inflation of a small balloon introduced into the œsophagus. It is evident that hyperacidity and hypersecretion do not necessarily cause symptoms and that symptoms formerly regarded as due to acidity can be induced without acid. These facts further support the current view that the majority of symptoms of indigestion are due to abnormal motility of the stomach and not to abnormal secretion.

Coronary Insufficiency Due to Acute Hæmorrhage.

ARTHUR M. MASTER AND HARRY L. JAFFE (*Journal of the Mount Sinai Hospital*, May-June, 1940) report a case of coronary insufficiency and myocardial necrosis due to acute hæmorrhage. The patient was a woman, aged nineteen years, who became pregnant five months prior to her admission to hospital, and during the third month of pregnancy developed bloody diarrhœa and fever for which sulphanilamide therapy was instituted without effect. She aborted two weeks prior to her admission to hospital, her temperature being 106° F., and her condition deteriorated so that on admission to hospital she was stuporous. Examination revealed extreme pallor of the skin and mucous membranes and numerous retinal hæmorrhages, but the heart was not enlarged. A systolic and diastolic murmur was present over the aortic area and was considered to be hæmic in origin; the systolic blood pressure was 130 millimetres of mercury and the diastolic pressure was zero; the pulse was of the Corrigan type. A blood count revealed 1,000,000 erythrocytes per cubic millimetre with a hæmoglobin value of 19%, 19,000 leucocytes per cubic millimetre of which 51% were polymorphonuclear neutrophile cells and 46% lymphocytes, and 320,000 plateiets per cubic millimetre. Sternal puncture revealed a very cellular bone marrow with a considerable increase of normoblastic activity; attempts at blood culture were without result; the blood urea figure was 49 milligrammes per 100 cubic centimetres of blood, and the electrocardiogram revealed a depressed *RS-T* segment with low *T* waves in the three standard leads and in the chest lead, indicating myocardial damage. The author's diagnosis was ulcerative colitis, with massive hæmorrhage, severe secondary anæmia and sulphanilamide intoxication. The patient's condition rapidly declined after admission to hospital and she died eight days later. The necropsy findings were acute ulcerative colitis of the entire colon and ileum, aspiration hæmorrhagic bronchopneumonic changes in both lungs and necrosis of the anterior and posterior papillary muscles; but the coronary arteries were normal. The authors state that the *RS-T* segment and *T* wave abnormalities in the electrocardiogram of this patient indicated acute damage to the heart muscle, which was confirmed *post mortem*. They are of the opinion that the myocardial changes may have been caused by the anæmia, by the infection or conceivably by the administration of sulphanilamide, although they have not noted that this drug has been shown to affect the heart. They state that necrosis limited to the papillary muscles is characteristic of interference with the coronary circulation, and in this case must be attributed to the extreme anæmia of rapid onset which produces coronary insufficiency with severe anoxæmia of the cardiac muscle, even in the presence of normal coronary arteries. The authors reported their case to demonstrate that myocardial infarction may occur without coronary artery occlusion.

Blood Protein Determinations.

THE routine determination of blood protein has been made possible by the new falling-drop method reported by Kagan. Having compared this method with that of chemical analysis, H. H. Shuman and Harold Jeghers (*The New England Journal of Medicine*, February 29, 1940) have estimated the blood protein level of 320 patients admitted to their medical service. Of the results, 75% were within the accepted normal limits of 6 to 8 grammes per 100 cubic centimetres, the albumin-globulin ratio varying from 1·5 to 2·5. Variations from the normal in the remaining 25% include hypoproteinæmia (a total less than 6 grammes per 100 cubic centimetres), hyperproteinæmia (a total over 8 grammes per 100 cubic centimetres), changes in the albumin-globulin ratio, and, rarely, the presence of abnormal protein (Bence-Jones). In this series there was only one case of hyperproteinæmia, the diagnosis being multiple myeloma. Other conditions associated with a high blood protein include dehydration, subacute bacterial endocarditis, metastatic bone lesions and specific infections, such as tuberculosis. A true estimate of the blood protein level in cases of dehydration cannot be obtained until the fluid balance has been restored to normal. The remainder of the abnormal group showed a lowered blood level. The chief causes of this finding were as follows: dietary deficiency, either low intake or poor absorption (carcinoma of the colon) or poor utilization (*diabetes mellitus*, acute and chronic infection), heart failure and hepatic disease. Œdema was a very common clinical finding in this group. A low blood protein level makes a survey of diet essential, and its determination is at present the easiest laboratory test available for detection of a dietary deficiency. In this series abnormal figures were found as commonly as significant deviations from the normal in such accepted routine procedures as examination of the urine and the making of blood counts. With regard to treatment, most persons with a hypoproteinæmia will respond favourably to dietary measures alone. Liver disease and gastro-intestinal disturbances will preclude this method of treatment and blood transfusion or the intravenous administration of amino-acids will be indicated. The importance of hypoproteinæmia in cardiac decompensation needs emphasis from the point of view of treatment.

Gastro-Intestinal Hæmorrhage.

D. H. KAUMP AND J. C. PARSONS (*The American Journal of Digestive Diseases and Nutrition*, May, 1940) discuss extrarenal azotæmia in gastro-intestinal hæmorrhage. They have found that patients with slight hæmorrhage show no abnormality in blood urea value, the average level being 26 milligrammes *per centum*. In severe hæmorrhage, when marked anæmia was present, with red cells about three million or less per cubic millimetre, a rise in the blood urea content was observed up to 90 milligrammes *per centum*. The chloride content of the blood and of the urine was low in a case reported by the authors; 419 and 495 milligrammes *per centum* represented the figures for blood chlorides on the second and third days after massive hæmorrhage. This was contrary to the observations of Borst, who found hyperchloræmia with low urinary chloride excretion. There was general agreement that the blood urea was increased most markedly on the third or fourth day after the hæmorrhage. The intravenous administration of normal saline solution was attended by clinical improvement, a falling of the level of urea in the blood and an increase in the level of blood chlorides.

Vitamin B_1 in Delirium Tremens.

HUGH E. KIENE *et alii* (*The Journal of the American Medical Association*, June 1, 1940) contend that the cause of *delirium tremens* is essentially a vitamin B_1 deficiency, which has been brought on by sudden deficits in the maintenance requirements; inadequate and irregular food and, at times, total abstinence from food is practically universal in acute *delirium tremens*. The delirium is an irritative stage of central (cerebral) neuronitis having its mechanism of function as a disturbance in the relationship of a damaged carbohydrate and vitamin B_1 metabolism. Ten patients admitted to hospital suffering from *delirium tremens* were divided into two groups and treated in the following manner. The first five were given whisky (32 ounces in twenty-four hours), synthetic vitamin B_1 intravenously (50 milligrammes or more in twenty-four hours) and high vitamin diet. The second group were given no alcohol nor vitamin by the intravenous route (except to one patient) and the same diet as the first group. The first five patients showed an immediate improvement in their physical and mental status, and in thirty-six hours visual hallucinations and fears were non-existent. The second group required an average of fifty hours before recovery. It is concluded that alcohol in the presence of large amounts of intravenously administered vitamin B_1 does not cause a continuation of the symptoms of *delirium tremens*; it was also observed that if adequate vitamin B_1 was given when alcohol was abruptly withdrawn, eventual recovery would also occur. The fact that recovery from acute symptoms occurred much more rapidly when vitamin was administered intravenously (even in the presence of continued drinking) would indicate an ætiology of primary vitamin B_1 deficiency in the presence of a deranged carbohydrate metabolism.

The Response to Treatment in Pernicious Anæmia.

M. C. RIDDLE (*The American Journal of the Medical Sciences*, August, 1940) has evolved an equation, which he suggests as a standard for measuring the relative effectiveness of treatment in pernicious anæmia: $I = 0{\cdot}78 - 0{\cdot}174E_0$, where I is the average weekly increase in the erythrocyte count after two weeks of treatment, and E_0 the erythrocyte count before treatment, expressed in millions of erythrocytes per cubic millimetre of blood. Observed values equal to or greater than those obtained from this equation indicate adequate treatment; observed values less than those calculated from the equation indicate inadequate treatment. The existence of various complicating factors, such as concurrent disease, transfusion or hæmorrhage, invalidates the use of this standard.

Special Articles on Psychiatry in General Practice.

(Contributed by request.)

XXIII.
SLEEP.

CERVANTES wrote:

> Now blessings light on him that first invented this same sleep: it covers a man all over, thoughts and all, like a cloak; it is meat for the hungry, drink for the thirsty, heat for the cold, and cold for the hot. It is the current coin that purchases all the pleasures of the world cheap; and it is the balance that sets the king and the shepherd, the fool and the wise man.

The psychiatrist realizes that Cervantes does not overestimate the importance of sleep. Since approximately a third of man's existence is passed in slumber, it would be surprising if it did not play a large part in his well-being. Insomnia is almost invariably an ætiological factor in the production of nervous and mental invalidism. Disturbance of the sleep rhythm is equally invariably a symptom.

The return to adequate sleep is the prelude to recovery. Without fear of contradiction, it may be said that sleep is a mind-saving tonic.

The problem of the production of sleep must be regarded from a wide angle. A brief analysis shows that the healthy person's slumber depends on a psychological state—tiredness and mental relaxation—and a physical state; again, there are tiredness and certain environmental conditions, such as a comfortable couch and freedom from external stimuli—light, sound, cold *et cetera*. This brief classification may be usefully extended as an *aide mémoire* in treatment.

The Psychological Aids.

Mental relaxation can be taught by explaining to the patient the technique of relaxing the body and limbs coincidentally with a sleep attitude of mind. Sleep must not be sought as such; it will arrive unbidden. Provided that relaxation is obtained, a slight amount of actual sleep will suffice the body needs. The main psychological treatment will of course deal with the solution of current worries; but there are certain specific worries which the physician must constantly bear in mind. Fear of death during sleep may be countered by a frank discussion of this topic. Fear associated with loneliness is often remedied by placing the patient in a ward, obtaining a night nurse, or even by the use of a night light.

Physical Aids.

Occupation, exercise and sport are obvious aids towards correct sleeping habits. A hot drink at bed-time allays gastric unrest. Cold feet need the assistance of the friendly hot-water bag. Adequate sleeping apparel and bed-clothes are important details. Any condition which produces discomfort or pain must be countered by appropriate measures.

Environmental Aids.

Environmental aids have been discussed above. We may add the quiet room, the restful surroundings and the quiet tactful nurse as desiderata.

But What Next?

It is an axiom that the more skilled the physician, the fewer the sedatives. Unfortunately even the most astute medical craftsman finds that sleep eludes the patient in spite of treatment along the above lines. The aid of the chemist has to be sought, since it is notorious that nervous patients often suffer from morbid insomnia. What is worse, ordinary doses of sedatives are frequently useless. As if the lot of the psychiatrist were not already hard enough, we must add immunity to standard doses of narcotics as a further contribution to the list of difficulties. The psychiatrist is forced to use heroic doses, and unless he does so he will usually fail.

The Choice of a Narcotic.

The chemist has produced such a galaxy of drugs that an adequate presentation would be the subject of a volume rather than a brief article. I have tried most of them, hopefully and enthusiastically, but with varying success, because people react differently to drugs. The factor of idiosyncrasy is ever present. There is no perfect and universal narcotic. The best advice is to rely principally on one drug, learn thoroughly its dosage and limitations. It is unwise to change prescriptions so constantly that one is master of none. The following brief comments may be helpful.

A soluble barbiturate has less "hang-over" than an insoluble variety. Barbitone soluble has therefore advantages over barbitone.

Potassium bromide and chloral are poor narcotics unless restlessness is the sole preventer of sleep.

Paraldehyde remains the most certain narcotic. Its vile taste is best disguised by syrup of orange. For patients undergoing "Cardiazol" therapy paraldehyde is invaluable, since the barbiturates are contraindicated.

Morphine should be prescribed only as a temporary measure and only in those cases in which pain is a factor. Frequently the homely aspirin or phenacetin alone or in combination will serve the same purpose. Amidopyrine employed cautiously is often useful.

For mild cases of the type produced by an "over-active mind" a phenobarbital in half-grain or one-grain doses may suffice. "Soneryl" has the advantage of a particularly mild "hang-over". Other proprietary barbiturates, such as "Amytal", "Sodium Amytal", "Nembutal", are too well known to need further description.

A combination of narcotics is often more successful than a single one.

A powder containing barbitone soluble, ten grains, phenobarbital sodium, three-quarters of a grain, calcium lactate, ten grains, may be usefully prescribed.

The Dosage of Hypnotics.

The golden rule of dosage is not the British Pharmacopœial standard, but the physiological effect. You must promise sleep, you must obtain sleep, you must use sufficient of the drug to achieve your objective. In this connexion it is psychological wisdom to give an adequate dose at the commencement rather than to step up gradually from minimal doses.

Faith is an essential ingredient of your prescription, and if at the outset you fail to redeem your promise, faith is lost. This has a bearing on the problem of addiction. The addict is produced partly by underdosage and partly by an ill-advised weaning process. Addiction may be countered by a preliminary conversation with the patient. He must be informed that the medicine is harmless, it is sure and will leave no habit. When the time for weaning arrives there is no suggestion that he must try to do without the medicine; instead he is told to have it by his bedside and if he needs it he can take it. Invariably he eventually finds himself forgetting to take it. The psychological mechanism is similar to that of the pocket handkerchief fetish. So long as it is with us we do not think of blowing the nose; but if we forget it, then anxiety ensues.

The Nomenclature of Drugs.

A word must be said as to the form of prescriptions. The employment of narcotics has been made more difficult by modern publicity. Press reports of suicides, addiction and therapeutics have made the public drug conscious. The pharmaceutical chemist involved in the coil of drug repression legislature often contributes to this by warning the patient to take his medicines sparingly. This combination of adverse influences necessitates care in the form of the prescription. It is wise to use names of unfamiliar significance, for example, barbitone instead of "Veronal", "K Br." instead of potassium bromide, tincture of thebaica for tincture of opium *et cetera*. In order to reassure the chemist, initial all doses which are greater than that which the pharmacopœia employs.

A Word to the Rightly Cautious.

Reference has been made to heroic doses. Such mental disorders as manias and confusional states may require unbelievable amounts of sedatives. Whenever possible these patients should be transferred to the hands of the practising psychiatrist. Their treatment is difficult even for those solely engaged in the specialty.

A dose of barbitone soluble, ten grains, is hardly heroic, yet in predisposed cases it may result in a worrying toxic reaction. The practitioner will attack this with confidence if he carries ampoules of picrotoxin, now issued with directions by the drug houses. Given intramuscularly, it is a safe antidote and has spectacular results. It will be seldom needed; but its possession, as with the handkerchief already mentioned, gives a feeiing of confidence.

Whenever toxic symptoms and signs appear—for example, coma, scarlatiniform rash, ulceration—the exhibition of barbiturates of every description must be discontinued. In this connexion the ubiquitous bromide mixture is a frequent

offender. Bromide intoxication as shown by drowsiness or ataxia, a feeling of "drunkenness", is quite common. It usually occurs in those who have a poor appetite. Limitation of bromides and the ingestion of additional sodium chloride rapidly produce a cure.

Conclusion.

The last word on the sleep problem has not been written; but at least we know that its production is vital to our success as physicians. It must be considered in every case of nervous and mental disorder. If not the *"fons et origo"*, it is the good companion of our therapeutic endeavours. As such we must give it the attention it deserves—always thought and occasionally courage tempered with discretion.

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Medical Societies.

MELBOURNE PÆDIATRIC SOCIETY.

A MEETING of the Melbourne Pædiatric Society was held at the Children's Hospital, Carlton, on June 12, 1940, DR. H. DOUGLAS STEPHENS, the President, in the chair. The meeting took the form of a series of clinical demonstrations by members of the society.

Bronchiectasis.

DR. RUSSELL HOWARD showed a boy, aged twelve years, suffering from bronchiectasis restricted to the lowest lobe of the right lung. Apart from an attack of measles at the age of three years, the boy had had no illness until late in 1939. On November 15, 1939, he was admitted to the Eye and Ear Hospital, suffering from sinus infection, and on December 7 right-sided endonasal antrostomy was performed. This was followed by consolidation at the base of the right lung associated with pyrexia, which was thought at the time to be due to pneumonia. On December 15, however, aspiration was performed and thick pus was obtained. The patient was then transferred to the Children's Hospital. He was very ill, the temperature reaching 104° F. The presence of pus at the base of the right lung was confirmed radiographically, and next day an attempt was made to drain the pus through an intercostal catheter inserted through a cannula, but no pus was withdrawn. A rib resection operation was undertaken and the pleural cavity was found to be empty and uninfected; but as the lung had been pierced by the trochar and contained pus, the opening was enlarged. A great deal of unhealthy lung tissue was exposed, honeycombed with pus-containing cavities, but no large single cavity was found; the smell was not offensive and a pure culture of hæmolytic streptococci was obtained from the pus. Subsequent to the operation a great quantity of pus was discharged along the drainage tube, and it soon became foul-smelling. An empyema cavity developed, which was drained at a higher point by an intercostal catheter connected with a continuous negative pressure system. Though a bronchial fistula resulted, the child's condition improved steadily. At the time of the meeting the lower wound had healed and the upper wound was reduced to a fine sinus communicating with a bronchus.

Dr. Howard explained that clubbing of the fingers was noticed for the first time one week after the original operation; it was impossible to be sure that the clubbing had not been present earlier. He showed from bronchograms the development of a gross degree of bronchiectasis restricted to the basal portion of the right lung, and said that scoliosis had occurred, though it had been almost eliminated by means of exercises and postural management. He went on to say that in spite of the unusual finding on culture of the pus, there was no doubt that the child had had a pulmonary abscess and that the bronchiectasis was probably a sequel to the abscess, though that fact could not be established beyond dispute on account of the previous occurrence of sinusitis and the likelihood of the preexistence of clubbing of the fingers. Dr. Howard said that if the bronchiectasis antedated the abscess there was no evidence that it was infected; the child had had no coughing. He also pointed out that the operation had necessarily to be performed in one stage because of perforation of the lung by the trochar and cannula. He mentioned that after the child had been allowed a period of convalescence he intended to perform lobectomy, because he considered that as the bronchiectasis was gross and limited in its distribution, operation rather than conservative treatment was distinctly indicated.

Subacute Recurrent Parotitis.

Dr. Howard also showed a girl, aged eight years, suffering from subacute recurrent parotitis. Over a period of four years intermittent swelling of the right parotid gland had occurred every two or three months, lasting from three days to three weeks. Dr. Howard said that the present attack had commenced ten weeks before the meeting, partially subsiding after three weeks; but it recurred rapidly after the girl sucked a lemon and it had persisted much longer than usual. The right parotid gland was swollen and was approximately the size of a pigeon's egg. The duct orifice was normal in appearance and he had been unable to express any mucopus from it. The girl's tonsils had been removed earlier in life and her teeth were in good order. He showed from X-ray films the absence of calculus and the presence of sialectasis.

Dr. Howard expressed the opinion that the condition was an example of subacute recurrent parotitis, probably due to recurrent inflammation of the main duct of the gland or of the finer ductules, and ascribed the sudden swelling after the eating of a lemon to the blocking of a large duct. He said that the condition was not uncommon, and patients had been shown at previous meetings of the society to illustrate it; they might or might not present sialectasis and the ætiology was shrouded in mystery. In discussing the treatment, Dr. Howard said that various methods of intermittent duct dilatation and injection of antiseptics into the gland had been suggested, but all seemed to him to be futile. All septic foci in the mouth should be removed. He thought the most rational treatment in the absence of gross infection was either avulsion of the auriculo-temporal nerve or X-ray therapy; both were effective measures for stopping the gland from secreting. He added that there was a tendency for the condition to clear up in the second or third decade of life.

DR. J. K. GARDNER showed another patient suffering from sialectasis, a Greek boy, aged nine years. At the age of two and a half years the right parotid gland became swollen and the swelling subsided after two or three weeks; but the condition recurred a year later and again when the boy was six years of age. At about that time he had had a compound depressed fracture of the skull with residual paresis of the left arm. A week before the meeting he had been admitted to hospital on account of a rheumatic relapse and at some previous time his heart had been affected. On his admission to hospital the right parotid gland was moderately swollen. Pus obtained from the duct orifice was examined and an attempt at culture was made; the pus contained phosphate crystals and a growth on culture media consisted of a mixture of cocci, diphtheroid bacilli and *Micrococcus catarrhalis*. Dr. Gardner showed from the skiagrams the absence of any radio-opaque calculus and the presence in a sialogram of definite sacculation throughout the gland substance. He stated that the swelling had subsided during the week, and commented that the sialographic technique appeared to have been useful as a form of treatment; the boy needed dental treatment and would probably benefit later from tonsillectomy. Dr. Gardner mentioned that he had been impressed by the absence of pain as a feature in the case.

DR. W. KENT HUGHES suggested the possibility that Dr. Howard's patient had a mixed parotid tumour; it had caused symptoms for years and was not obstructive. He remarked that the other patients had red inflammation of the duct orifice in the mouth, and he thought the condition would clear up after antiseptic irrigation of the duct.

DR. E. E. PRICE said that he was interested in the matter under discussion. He had only had three patients with the same complaint in his care; but he had noted somewhat different features. He agreed that in each of the cases discussed that night the ducts were dilated, and thought that recent canalization accounted for the redness at the orifice to which Dr. Kent Hughes had referred. In his own cases Dr. Price had been impressed by the patulous state of the duct orifice; one child could insufflate air and produce froth at will. Dr. Price had formed the impression that a neuro-muscular rather than an obstructive factor was at work. Another point of difference was that the swollen parotid glands of his patients felt like rubber and were not nearly so hard as was that of Dr. Howard's patient. If the hardness in the latter case was due to obstruction at all, the obstructive element might be overcome by the passage of sounds suitably graduated in size. Dr. Price

added that one of his own patients had recovered after dental extractions, tonsillectomy and postprandial massage of the affected gland.

DR. J. L. FREW said that there were always some patients at the Royal Melbourne Hospital undergoing treatment for intermittent swelling of the parotid gland. He expressed the view that they should not discard the diagnosis of sialectasis on the score of hardness of the swelling.

DR. H. DOUGLAS STEPHENS asked Dr. Howard whether he had seen any bilateral cases; Dr. Stephens also asked for the differential diagnosis from Mikulicz's disease.

Dr. Howard said that subacute recurrent parotitis was a definite entity and that he had seen three examples in Melbourne, of which one was bilateral. He believed that at times in the same patient there was an alternation of swellings of the parotid glands, and even the submaxillary glands might be involved. He was satisfied that in the case discussed that night the pathological process was not a mixed tumour of the parotid; he based that opinion on the observed facts that the tumour varied grossly in size from time to time and had been enlarged to double the previous size quite rapidly after the patient had eaten a lemon. He said that it was generally conceded that the main duct was obstructed by inflammation and that the obstruction led to sialectasis; the hardness of the gland varied considerably with the tension. It was held by some observers that the condition resulted from oral infection; but Dr. Howard was confident that that factor could be dismissed in the case under discussion. Another view was that anaphylaxis occurred; the evidence for that was the presence of masses of eosinophilic cells in the secretion "milked" from the duct orifice, the intermittent nature of the swellings and their definite tendency to spontaneous subsidence.

Dr. Howard then discussed the question of treatment. He said that there was every reason to expect recovery as age progressed; one of his previous patients had had no swellings for several years. The methods of treatment usually adopted were intermittent dilatation of the duct with whale-bone bougies and injection of antiseptics into the gland; but he had not been impressed with the effectiveness of those procedures. The girl he had shown that night had had the swelling for twelve weeks, and he could find no evidence of obstruction at or near the orifice of the duct; it was difficult to dilate the duct, which became more deeply situated in the gland itself. He had cause to remember a case of stenosis of the mouth of the duct following upon surgical interference in an attempt to overcome the obstruction. He had reached the conclusion that as the disease process was unilateral and had been going on for years, it was reasonable to stop the secretion of the affected gland; and he intended to avulse the auriculotemporal nerve. He had been put right off the idea of puncturing the parotid gland in several places by a previous experience; the gland became considerably more enlarged almost immediately and the end result was not satisfactory. In conclusion, Dr. Howard said that the boy he had shown suffering from bronchiectasis was an ideal candidate for lobectomy, and he hoped that he would have a later opportunity to report on the progress of both patients.

Therapeutic Use of Spinal Anæsthesia.

DR. D. O. BROWN said that he wished to present the relevant features of four case histories with the object of indicating the possible value of spinal anæsthesia in treatment. He was indebted to Dr. Gardner for directing his attention to an article published in *The British Medical Journal* of December 23, 1939, at pages 1224 and 1225. The article was by E. D. Telford and H. T. Simmons, and was entitled "Treatment of Gastro-Intestinal Achalasia"; in the article they claimed that seven children with megacolon had been treated by spinal anæsthesia alone with excellent results, and one with cardiospasm had been treated and had remained completely cured. It had been observed that if children suffering from gastric achalasia were submitted to spinal anæsthesia they were so much better afterwards that there was no need to carry out an operation on the sympathetic nervous system.

Dr. Brown then proceeded to give an account of his experience of the subject, which he admitted was limited to a small series of cases. He said that he was showing the patients before the members of the society prematurely, as he was relinquishing practice for military service abroad at an early date. He did not wish to make any extravagant claims and would merely give the facts without attempting to explain them; he did not think there was a rational explanation as to why the effect of the spinal anæsthesia was durable.

The first patient of the series was a child with congenital diverticula of the bladder associated with right-sided hydroureter and hydronephrosis, whom he had shown previously at a meeting of the society on April 10, 1940 (THE MEDICAL JOURNAL OF AUSTRALIA, June 22, 1940, page 876). Dr. Brown had operated on the boy, using spinal anæsthesia. There had been some discussion as to whether the ureter would have to be implanted; but at the operation he had not found it possible to remove the diverticulum without endangering the ureter, and he had divided it and implanted it into the bladder. He was glad to be able to report that from the technical point of view he had encountered no difficulties and that the patient had made very satisfactory progress; he passed urine in normal fashion and his general condition from the clinical point of view was very good; but Dr. Brown had not obtained a pyelogram since the operation.

The next patient in the series was a baby, who at the age of four days had been admitted to hospital with the signs of acute intestinal obstruction; the baby was vomiting everything given, the abdomen was very distended and the bowels had not been opened. The presumptive diagnosis was that the signs were due to congenital absence of a section of small bowel; but as it was thought that they might prove to be due to neuro-muscular incoordination, Dr. Brown had decided to give preliminary spinal anæsthesia before opening the abdomen. He introduced one cubic centimetre of "Percaine" solution into the theca, and the effect was admirable. The interesting thing was that about thirty seconds after the introduction of the spinal anæsthetic the fæces came out of the rectum explosively and the abdomen went down flat; it was a most striking happening. Dr. Brown had subsequently opened the abdomen and found that a segment consisting of the caecum and ascending colon was tense and bluish and showed no evidence of haustra; its condition resembled that seen at times in intussusception; but he could find no organic obstruction. After the operation they had had a great deal of trouble with the wound on account of distension of the abdomen; injections of pituitrin had not helped, but an excellent response was obtained to an injection of 0·25 cubic centimetre of acetylcholine. The baby had made a good recovery and was aged one month at the time of the meeting. In commenting on the case Dr. Brown said that death in early infancy was the rule in the presence of that syndrome, and at times no satisfactory explanation was discovered at autopsy. The condition was probably Hirschsprung's megacolon in the incipient phase. He had the impression that the patient would have died if the spinal anæsthetic had not been used. He strongly advocated spinal anæsthesia for very young babies with acute intestinal obstruction.

Dr. Brown then gave an account of how it had been discovered that a boy undergoing treatment for Perthes's disease at the orthopædic branch of the hospital at Frankston had been found to have gross bilateral hydronephrosis and hydroureter with a tendency to distension of the bladder. Though he had regarded the condition as neuro-muscular in origin, he had performed suprapubic cystostomy as a life-saving procedure, and at that time by retrograde catheterization he had satisfied himself that there was no urethral obstruction. As subsequent treatment presacral neurectomy or ganglionectomy was under consideration; but he had decided to give the boy spinal anæsthesia as an alternative method of treatment. At that stage Dr. Brown explained that for spinal anæsthesia he used a 1 in 1,500 solution of "Percaine" and had worked out the dosage roughly in the following way: for children aged one month, one cubic centimetre; six months, two cubic centimetres; one year, three cubic centimetres; three years, four cubic centimetres; six years, five cubic centimetres; and nine years, eight cubic centimetres. On the occasion to which he had referred, a good anæsthetic effect was obtained and he had obtained on the day before the meeting a pyelogram which was perfectly normal in appearance. Dr. Brown made it plain that he did not claim that that finding was due positively to the spinal anæsthesia; it might have been the result of suprapubic cystostomy. He had, however, failed to find any sign of organic obstruction, and he wished to record the suggestion that in spinal anæsthesia they might have a hope of effective treatment for what was a truly dreadful condition.

The last patient in the series was a boy who had been transferred some five years earlier to the Children's Hospital from a general hospital with the diagnosis of abdominal tuberculosis or malignant disease. Dr. Brown said that the boy was pale and miserable; the abdomen was considerably enlarged and many rocky hard masses could be felt. On laparotomy it was found that the intestine was packed full of fæcal masses and the bladder was dilated up to the level of the xiphisternum. The bladder was emptied by catheter drainage and the fæces were evacuated from the bowel. From time to time it had been necessary to disimpact fæcal

accretions. The boy had a big dilated colon, but not a true Hirschsprung's megacolon. No further bladder trouble had been manifested. On June 1, 1940, Dr. Brown gave the patient spinal anæsthesia, and though it did not work particularly well, at least he could report that the boy had not needed an enema since and that the bowel actions were much easier and more regular.

DR. J. W. GRIEVE congratulated Dr. Brown on what he considered to be a very important communication to the society. Dr. Grieve had himself been particularly impressed with neonatal gastro-intestinal dysfunction as a problem; occasionally a spontaneous recovery occurred; but it was most useful and informative to have a practicable method of dealing with it. The presence of hydroureters was also regarded as a hopeless problem, and the result Dr. Brown had described was of distinct promise.

DR. C. H. OSBORN added his congratulations and asked whether the type of gastro-intestinal spasm to which Dr. Brown had referred was restricted to the neonatal period; he wished to know whether the same sort of thing was liable to occur later on in life.

DR. E. E. PRICE said that Dr. Brown had introduced a subject which would stimulate thought and action. More and more evidence was accumulating on the efficacy of spinal anæsthesia in therapy, and Dr. Price had also wondered why the effect should be permanent. In an attempt to contribute to the answering of Dr. Osborn's question, Dr. Price mentioned that he had performed, at the Royal Melbourne Hospital, an abdominal section on a man showing signs of intestinal obstruction; he had found nothing to account for the obstruction, but the patient had made a satisfactory recovery.

DR. H. DOUGLAS STEPHENS recalled some of the details of the case of a baby, aged four and a half months, suffering from intestinal obstruction and enlargement of the abdomen; no result had been obtained on the passage of a small catheter *per rectum*, and at operation he had found a huge hydroureter on the left side, which in its course through the pelvis had blocked the sigmoid colon and rectum. He had removed the kidney and ureter and the child had recovered. Dr. Brown's communication had caused Dr. Stephens to wonder whether he could have relieved the child by inducing spinal anæsthesia; but as the ureter was so large he was doubtful whether relief would have been obtained without operation. He regretted that Dr. Brown had left the meeting and was unable to reply to the discussion.

Traumatic Meningocele.

DR. HOWARD WILLIAMS, on behalf of DR. J. G. WHITAKER, showed a girl, aged four years, with a traumatic meningocele. At the age of one year and seven months the child had fallen and hurt the back of her head. The accident had caused no special concern for two weeks, but at that stage, on account of irritability and persistence of the swelling, she had come under observation at the hospital. A large subaponeurotic hæmatoma was present, and from the skiagrams it was learned that she had sustained an occipital fracture. After being in the hospital for several weeks she was returned to her home with directions to rest in bed and report at the out-patient department within six weeks. Those directions were carried out, and when she visited the out-patient department it was found that there was an extensive deficiency of bone around the occiput, through which a large soft pulsatile swelling could be felt. The condition was quite symptomless, but the tumour had gradually increased in size at first, though the size had remained stationary for the past two years.

DR. W. KENT HUGHES postulated that the interparietal bone had either never developed or had been absorbed as a result of the trauma.

DR. MOSTYN L. POWELL, while admitting from the information given that trauma had had some influence and that the original swelling must have contained blood, expressed the view that there must have been a congenital defect of bone to produce the condition present at the time of the meeting. The blood cyst would be subdural but extraarachnoidal, and the defect had acted as a safety valve for the increased intracranial pressure, avoiding the necessity for an operation; but the patient's bone had continued to be eroded with absorption of metabolites. He added that he had tried roughly and quickly to gauge the normality of the fields of vision, and thought that she might have some deficiency in the right field.

DR. H. DOUGLAS STEPHENS also thought there must have been a congenital basis for the condition; from the site of the hole in the bone there might have been a Wormian bone present at the time of the accident. He remembered having seen a baby, aged nine days, with a hole around the margin of the posterior occipital protuberance which was altogether separate from the posterior and the anterior fontanelles; if that child had fallen on the head he could conceive the development of a similar condition to that in Dr. Williams's case.

Congenital Shortening of the Œsophagus.

DR. J. W. GRIEVE showed a male patient, aged nine months, with congenital shortening of the œsophagus. The birth weight was nine pounds three ounces and the birth was attended by no special difficulty. The baby was naturally fed for one month and then on dried cow's milk. He had gained six or seven ounces in weight each week until he reached the age of four months, when vomiting commenced. The vomitus was small in amount and had the appearance of containing coffee-grounds. That type of vomiting had continued and was increasing both in frequency and in amount. After a severe vomiting attack the baby's stools would be black for several days. He had gained only five ounces altogether in weight in ten weeks. He was restless and cried and threw himself about before a vomiting turn, but seemed to be relieved when he had vomited. On investigation it was found that 85% of the leucocytes were lymphocytes; the hæmoglobin value was estimated at 85%; radiographic examination while the child was swallowing barium revealed that he had congenital shortening of the œsophagus and that the stomach was partially intrathoracic. An œsophagoscopic examination revealed a change from œsophageal to gastric mucosa seven inches from the alveolar margin of the jaw, but no definite cardiac sphincter was seen; the œsophagus sloped away to the right instead of to the left, and though there was no alteration, slight oozing from the gastric mucosa was seen through the œsophagoscope. Dr. Grieve also reported the results of an alcohol test meal; after half an hour the total chlorides were estimated at 60 and the combined acidity at 5 units; at the end of one hour the total chlorides were 60 and the combined acidity 12; at the end of one and a half hours the total chlorides were 70 and the combined acidity 15 units; and no free hydrochloric acid was present. Dr. Grieve went on to say that the baby had vomited mucus containing dark blood on several occasions while in hospital. The child's weight on his admission was sixteen pounds twelve ounces, and it had fallen to fifteen pounds three ounces; he was being fed every two hours with a sweetened cow's milk mixture (usually five ounces of a mixture of three parts of milk and one part of water), and also some broth and custard.

Dr. Grieve said that the condition was frequently not discovered until the patient was considerably older than the one under discussion. He directed attention to an article in *The Journal of Pediatrics* of January, 1936, in which the radiological criteria for the diagnosis were described, and also to an interesting article by T. P. Dunhill in *The British Journal of Surgery*, Volume XXII, 1934, page 475.

DR. JOHN D. BEGG suggested that the stomach was herniated back through the diaphragm and the œsophagus was not necessarily shortened.

DR. A. GWYNNE VILLIERS said that she had come across a similar case at the Queen Victoria Hospital. That patient had vomited coffee-ground material and had had cardiospasm, and the radiological picture was of the same nature as in the present case; the child at the age of four years was having only occasional vomiting attacks at increasingly long intervals.

DR. RUSSELL HOWARD said that there appeared to be no obstruction, and suggested that it might be instructive to make another radiographic examination by means of a barium meal with the patient in the Trendelenburg position.

DR. H. DOUGLAS STEPHENS said that after a study of the skiagrams shown by Dr. Grieve he thought that there was a shortening of the œsophagus rather than a paraœsophageal pouch.

Thyreotoxicosis.

Dr. Grieve also showed a girl, aged nine years, who had been under treatment for a year for thyreotoxicosis. She was over-weight and had an enlargement of the thyreoid gland, and over a period of three months she had been given some Lugol's solution regularly. She had become toxæmic and her condition was becoming worse when she was admitted to hospital with exophthalmos, goitre and chorea. Dr. Grieve had realized that the condition was more severe than usual and would probably require surgical treatment. The basal metabolic rate was +35%. The child had had German measles followed by catarrhal jaundice, but since then she had gained in weight and was looking rather better. The pulse rate varied between 100 and 120

beats per minute; she was still thyreotoxæmic and goitrous, but the choreiform tremor had disappeared. Dr. Grieve had consulted the surgeons, who were ready to operate or to use deep X-ray therapy, and he was prepared to hand the child over to Dr. Whitaker for future management. Dr. Grieve said that he was glad to see Dr. Frew present and would like to hear his opinion of the case.

DR. J. L. FREW said that he was interested in the treatment of the condition in young subjects and that there was a school of thought in favour of deep X-ray therapy. He regarded the condition of the patient shown by Dr. Grieve as an example of the fairly toxæmic group; but by deep X-ray therapy good results were obtained in patients with basal metabolic rates up to +50%. Dr. Frew had come to the meeting to try to find out whether deep X-ray therapy was in favour at the Children's Hospital.

DR. D. O. BROWN said that he was interested in the patient's case. He had thought that surgical intervention was indicated, but was glad to hear Dr. Frew's support for deep X-ray therapy. He advocated that form of treatment for the patient; if operation became necessary later it would not be so difficult.

DR. M. O. KENT-HUGHES said that he had come across a patient, aged twenty years, who had obtained a good result from deep X-ray therapy for toxic goitre. He also knew an older patient for whom the method had not been successful, but the old technique had been followed. For the past five years a newer technique had been available and he believed that the results were excellent.

DR. RUSSELL HOWARD said that when he was abroad he had formed the view that deep X-ray therapy was advocated for young subjects, and when the condition recurred after surgical treatment. Experiences at operation, however, when portions of thyreoid tissue were discovered to be deeply situated and larger than anyone would have thought, suggested that in those cases at all events deep X-ray therapy could not be expected to give better results than good surgical measures.

Dr. Grieve, in reply, thanked those who had taken part in the discussion. He also said that he would talk the matter over with Dr. Whitaker, and it was probable that they would undertake deep X-ray therapy for the patient.

THE MEDICAL SCIENCES CLUB OF SOUTH AUSTRALIA.

A MEETING of the Medical Sciences Club of South Australia was held at the University of Adelaide on August 2, 1940.

Post-Mortem Findings in Cardiac Infarction and Gall-Stones.

PROFESSOR J. B. CLELAND summarized the results of a series of autopsies carried out at the Royal Adelaide Hospital in cases of cardiac infarction and gall-stones. He said that 4,000 tabulated post-mortem results obtained between the years 1920 and 1939 were available for consideration; the period of each 1,000 post-mortem examinations covered about five years. From the results that he would give later, it appeared that during the last five-year period the incidence of cardiac infarction had increased, and the increase was not due merely to an ageing population. The cause was at present obscure. The cases of myocardial infarction in successive thousands were: 13, 5, 11, 28—a total of 57, or 1·4%; the cases of myocardial fibrosis in successive thousands were: 3, 6, 11, 9—a total of 29, or 7%. The ages of the subjects of the last 2,000 autopsies were, for males: forty to forty-nine years, 1; fifty to fifty-nine years, 5; sixty to sixty-nine years, 10; seventy to seventy-nine years, 5; eighty years and over, 1; this made a total of 22 males. For females the ages were: forty to forty-nine years, 1; fifty to fifty-nine years, 4; sixty to sixty-nine years, 7; seventy to seventy-nine years, 4; eighty years and over, 1; this made a total of 17 females. In the first, second, third and fourth (930) thousand autopsies the numbers of persons who were aged fifty years or over were 519, 509, 572 and 564 respectively. Though the persons aged over fifty years were approximately the same in the third series (572) and the fourth series (564), the numbers of cardiac infarctions were 11 and 28. This was not due to any appreciable increase of age, as was shown by the following figures: in the third series 192 persons were aged fifty to fifty-nine years, and in the fourth series 199; in the third series 206 persons were aged sixty to sixty-nine years, and in the fourth 193; in the third series 142 persons were aged seventy to seventy-nine years, and in the fourth 135; in the third series 32 persons were aged over eighty years, and in the fourth 37.

With regard to gall-stones, Professor Cleland said that the tabulated results of 3,000 autopsies at the Royal Adelaide Hospital showed that gall-stones were present in 302 subjects; 158 were males and 144 females. The gall-stones included all types. It would be seen that gall-stones were much more frequent in women, as there were twice as many male subjects as female, and that their incidence increased regularly over the decennial periods. It was probable, for instance, that in the age period sixty to sixty-nine years one in every ten men had gall-stones, and one in every 4·5 women. It was important to bear this frequency in mind in the analysis of the symptoms, as in the vast majority of cases the gall-stones were presumably "silent". Of 302 examples in 3,000 autopsies, 158 were in men and 144 in women. The following was the age distribution:

Males.

20-29	4 out of 151	= 1 in	37·7
30-39	4 out of 200	= 1 in	50·0
40-49	22 out of 299	= 1 in	13·6
50-59	26 out of 355	= 1 in	13·6
60-69	45 out of 440	= 1 in	9·8
70-79	43 out of 302	= 1 in	7·0
80 and over	14 out of 74	= 1 in	5·0

Age not stated, 70.
Youngest 27, oldest 90.

Females.

20-29	6 out of 148	= 1 in	24·6
30-39	17 out of 161	= 1 in	9·5
40-49	18 out of 154	= 1 in	8·5
50-59	26 out of 168	= 1 in	6·5
60-69	36 out of 163	= 1 in	4·5
70-79	28 out of 71	= 1 in	2·5
80 and over	11 out of 26	= 1 in	2·4

Elderly, 1.
Age not stated, 1.
Youngest 25, oldest 90.

The ratio of male to female subjects in the total number of post-mortem examinations was 2 : 1; but the numbers of gall-stones were nearly equal. There were 158 instances of gall-stones in 2,005 men—one in 12·6; there were 144 instances of gall-stones in 989 women—one in 6·9. In 53 of the 302 cases carcinoma (or in one instance sarcoma) was present—one in six (of carcinoma). There were 2,144 subjects aged over forty years, of whom 271 had gall-stones—one in eight.

Special Correspondence.

LONDON LETTER.

BY OUR SPECIAL CORRESPONDENT.

THESE are difficult, nay, almost impossible times for correspondents of all classes, military, medical and just personal. There is much to be said, but it cannot be said. News must be read between the lines, rather than in them. In May, 1935, there was published the sixth edition of a book by Harold P. Clunn, entitled "The Face of London", and dedicated to the "industry and enterprise of the citizens of London". It may be some little time before the seventh edition appears, but if and when it ever does, its title will have to be altered to the "Changed Face of London" and the dedication will be that which the non-Hitlerian world has already offered to the courage and indomitable heroism of the citizens, residents and visitors of and to this great bastion of freedom.

Another strange sign of the times, strange at least to all members of the medical profession, is a letter addressed by the Chairman of Council of the British Medical Association to all whom it may concern. He writes: "It is very evident that as the war goes on cases of serious financial distress will arise among members of our profession. The practices of some have disappeared entirely owing to evacuation, others have taken on military duties which involve them in heavy financial loss . . . It would therefore seem right that the medical profession should itself come to the assistance of its less fortunate members." This letter was written on August 20, 1940. Had it been circulated a month later its terms would have been even more appealing, and for reasons which even the most unobservant of visitors to the medical Mecca of Central London will immediately see for himself.

War causes many disruptions of, and curious alterations in, our ordinary peace-time activities. The British Medical Association House no longer seethes with the activities of Council, committees, and visitors from home and overseas. The affairs of the Association are now guided, not by the Council, but by an emergency Central War Executive Committee. *The British Medical Journal*, being regarded by the National Government as an essential war-time necessity, carries on under its board of directors and its staffs, though there are paper and other difficulties of considerable magnitude which may yet necessitate some drastic alterations in mode and place of publication.

Yet with it all, and notwithstanding the troglodytic existence most of us now lead, medicine and the country carry on with that bulldog tenacity so characteristic of the breed both at home and overseas.

Correspondence.

THE USE OF HISTAMINE IN THE TREATMENT OF NEURO-VASCULAR HEADACHE.

SIR: Referring to a letter by Dr. D. L. Barlow in the correspondence section of THE MEDICAL JOURNAL OF AUSTRALIA on October 26, 1940, I regret not having made myself sufficiently clear in the above report on October 5, 1940. Of course I recognize allergy as a factor in the production of certain types of headache. It was not intended in that report to enter into a detailed discussion of the various causes of headache. It was merely intended to discuss the use of histamine in the treatment of a definite group of headache syndromes in which there was no detectable extracranial or intracranial pathological lesion. It was emphasized that only when all possibility of a pathological lesion had been excluded should a neuro-vascular mechanism be considered. Should a condition such as allergy, focal sepsis, intestinal disorders, or a lesion of the ears, eyes, nose or throat *et cetera* be detected, it must be treated before the patient is subjected to a series of histamine injections.

Yours, etc.,

W. LISTER REID.

Sydney,
November 24, 1940.

POST-WAR ORGANIZATION.

SIR: That a new world order will follow this war is certain. What that order will be, no one at present can predict with any degree of certainty, other than that there will be a translation of men and money, as a result of which, what of the social, political and economic upheaval that will follow in its train? Motives and ideals are not enough; but it is possible to observe certain trends which can be studied systematically and objectively, even if the world is on fire. Let us, at this stage, by clear thinking and action, make a beginning to observe these trends, and if possible, may we be able to see their roots and translate our solutions into a better understanding, a better realization of misery and suffering, and a better way of living against the piling up of the methods of destruction and the fictitious values of men and money.

Does all this affect us, the members of our profession? In my opinion very intimately. Are we not the members of a supposedly educated class, the majority of whom make close personal contact with many families and in whose powers it is possible to observe these trends? It is my experience that it is more possible to obtain a truer idea of a man's thoughts in the precincts of his own home rather than in the rush and bustle of business life. (With tact and discretion, without being political.) Many may answer that it is not our business to make these inquiries. My regret is that there is not within our profession a committee of social studies, so that we might correlate these thoughts, not only for our professional benefit, but in the wider sense of preventive and social medicine.

The thin edge of the wedge of the socialization of our profession already has been driven in. In the post-war years that same wedge will undoubtedly be driven in further, so that it is our duty now to give every consideration to existing factors and to try to anticipate any splitting of the timbers of our profession, so that it will be done with benefit and justice to ourselves, as well as to those whom we may serve.

Observe the events since the last war and you will see that totalitarianism is well on the way, modified or otherwise, in this country, to date lightly touched by it or the horrors of war; but nevertheless, at no time in the world's affairs is greater clarity of thought and action required than at the present time.

If it is possible for us to be freed from bias, then let our profession make a contribution that is on a plane equal to those things for which we are fighting.

Yours, etc.,

KENNETH ADDISON.

Sydney,
November 25, 1940.

THE RADIOLOGICAL DIAGNOSIS OF TUBERCULOSIS.

SIR: I have been surprised by the manner in which some enthusiastic radiologists and physicians (with slight radiological training) make a rapid diagnosis of active or healed tuberculosis from the 35-millimetre film.

At a recent meeting at the British Medical Association House, and in Lieutenant-Colonel Cooper's recent article in *The British Medical Journal*, references were made to the state of activity of lesions shown in the small film.

After thirty-two years of X-ray experience, I still find it very difficult to decide whether the lesion is active or quiescent even where a good quality seventeen-inch by fourteen-inch film is available.

A good guess can be made, but auscultation, sputum and other tests should be considered as well.

Further, one frequently sees small areas of consolidation in the upper lobes in the course of an ordinary "influenza". These patches are almost always labelled as tuberculous, but if a further examination is made six weeks later they will have completely disappeared.

I think it would be wise to follow the overseas experts and consider the miniature film as one showing "gross pathology" and trust to the larger film to show "detailed pathology"; the question of activity might be left until all clinica data are available for consideration and not decided on the say-so of a radiologist after the examination of a single film.

Yours, etc.,

J. G. EDWARDS, M.B., Ch.M.

185, Macquarie Street,
Sydney,
November 27, 1940.

INFECTION BY EPIDERMOPHYTON INTERDIGITALE.

SIR: Recently I have heard, casually, two bitter complaints about the trials of infection with the fungus *Epidermophyton interdigitale*. One complaint was made by a trainee in a Royal Australian Air Force school. With the onset of summer complaints will probably be frequent, and medical officers may be glad to know of a simple method of relieving the sufferers.

Cleanliness is the first essential for prevention, and dryness the second. Treatment, when prevention has failed, is to apply some softened soap to the affected surface after it has been thoroughly washed with soap and water, as often as possible.

I have tried most remedies and have not found a cure, but can recommend soap for controlling the trouble.

Yours, etc.,

W. G. HEASLIP.

Institute of Medical and Veterinary Science,
Adelaide,
November 28, 1940.

Post-Graduate Work.

RESIDENTIAL COURSE IN OBSTETRICS AT SYDNEY.

THE New South Wales Post-Graduate Committee in Medicine announces that a residential post-graduate course in obstetrics will be held at the Royal Hospital for Women, Paddington, from January 5 to 11, 1941. The fee for the course is five guineas, and membership is limited to five. Applications for registration, accompanied by a remittance

for the amount of the fee, must be made to the Secretary, New South Wales Post-Graduate Committee in Medicine, the Prince Henry Hospital, Little Bay, from whom copies of the programme of the course will shortly be available.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 238, of November 28, 1940.

ROYAL AUSTRALIAN AIR FORCE.

CITIZEN AIR FORCE.

Medical Branch.

Alfred Ladyman Tostevin, M.B., B.S., D.O. (Oxon), F.R.A.C.S., is granted a commission on probation with the temporary rank of Squadron Leader, with effect from 14th October, 1940.

The following are granted commissions on probation with the rank of Flight Lieutenant, with effect from 7th October, 1940: Robert Murray Kavanaugh and Norman Towning Wood.

Flight Lieutenant S. K. Wilson is transferred from the Reserve to the Active List, with effect from 7th October, 1940.

Squadron Leader W. D. L. Farrar, M.B., B.S., is granted the acting rank of Wing Commander, with effect from 1st October, 1940.—(Ex. Min. No. 81—Approved 20th November, 1940.)

RESERVE.

Medical Branch.

Colin Hector Anderson, M.B., B.S., is granted a commission on probation with the rank of Flight Lieutenant, with effect from 14th October, 1940.—(Ex. Min. No. 82—Approved 20th November, 1940.)

Notice.

THE next library seminar arranged by the Post-Graduate Directors of Medicine, Surgery and Pathology will be held at the Prince Henry Hospital, Little Bay, on Monday, December 9, 1940, at 4.30 p.m. The subjects will be: (*a*) closed drainage of empyema with and without rib resection, (*b*) plasma *versus* serum in the treatment of hæmorrhage and shock. A cordial invitation to be present is extended to all medical practitioners.

The holding of the monthly library seminars and clinico-pathological conferences at the Prince Henry Hospital will be discontinued until February 10 and 24, 1941, respectively, when they will be resumed again for the remainder of the year.

Obituary.

ANNIE LISTER BENNETT.

WE regret to announce the death of Dr. Annie Lister Bennett, which occurred on October 24, 1940, at Mooroopna, Victoria.

CHARLES ALEXANDER CROTHERS.

WE regret to announce the death of Dr. Charles Alexander Crothers, which occurred on November 29, 1940, at North Sydney, New South Wales.

JOHN ALEXANDER NEWELL.

WE regret to announce the death of Dr. John Alexander Newell, which occurred on December 2, 1940, at Malvern, Victoria.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Gray, Franklin John, M.B., B.S., 1939 (Univ. Sydney), Sydney Hospital, Sydney.

Rychter, Szyja, M.D., 1936 (Paris); having passed the examination prescribed by the University of Sydney under Section 4, subsection (3), of the *Medical Practitioners Act*, 1912, of New South Wales, as amended by Act No. 33, 1915; 18, Georgina Street, Newtown.

Diary for the Month.

DEC. 10.—New South Wales Branch, B.M.A.: Ethics Committee.
DEC. 10.—Tasmanian Branch, B.M.A.: Branch.
DEC. 12.—New South Wales Branch, B.M.A.: Branch.
DEC. 13.—Queensland Branch, B.M.A.: Annual Meeting.
DEC. 17.—New South Wales Branch, B.M.A.: Medical Politics Committee.
DEC. 20.—Queensland Branch, B.M.A.: Council.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia.

Editorial Notices.

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